

## **Exhibit 12 – Expert Report of Dr. Goodman**

**Expert Report of  
Julie E. Goodman, Ph.D., DABT, FACE, ATS**

**In re: New-Indy Emissions Litigation**

Prepared by



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## ***Abbreviations***

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ATS	Academy of Toxicological Sciences
ATSDR	Agency for Toxic Substances and Disease Registry
CalOEHHA	California Office of Environmental Health Hazard Assessment
COVID-19	Coronavirus Disease 2019
IME	Independent Medical Examination
LOAEC	Lowest Observed Adverse Effect Concentration
LOAEL	Lowest Observed Adverse Effect Level
MoA	Mode of Action
MRL	Minimum Risk Level
NIOSH	National Institute for Occupational Safety and Health
NOAEC	No Observed Adverse Effect Concentration
NOAEL	No Observed Adverse Effect Level
OEL	Occupational Exposure Level
OSHA	Occupational Safety and Health Administration
ppb	Parts Per Billion
RfC	Reference Concentration
RfD	Reference Dose
TRS	Total Reduced Sulfur
UF	Uncertainty Factor
US EPA	United States Environmental Protection Agency
US	United States

# 1 Overview

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This report was prepared at the request of Morgan, Lewis & Bockius LLP in the context of the New-Indy Emissions Litigation. The Plaintiffs in this case are current or former residents of the community near the New-Indy Catawba LLC (hereinafter, "New-Indy") paper mill. The Plaintiffs claim to have developed various health effects from exposures to odorous gases originating from the New-Indy paper mill. Specifically, the Plaintiffs claim that they were exposed to total reduced sulfur (TRS) compounds, in particular hydrogen sulfide and methyl mercaptan, and that these exposures occurred at their properties in the community near the paper mill.

I evaluated whether it can be concluded to a reasonable degree of scientific certainty that Plaintiffs' alleged health effects were attributable to exposures to odorous substances (specifically, hydrogen sulfide and methyl mercaptan) that allegedly emanated from the New-Indy paper mill. I also critically reviewed the reports of Plaintiffs' experts, Dr. Allison Hecht, Dr. Deborah Barsotti, Dr. Harold Palevsky, Dr. William Fee, and Dr. William Meggs.

My opinions, summarized below, are based on a review of a substantial number of case-specific, scientific, and regulatory documents, as well as my training and experience in toxicology, epidemiology, and risk assessment.

## 1.1 Health Effects of the Chemicals of Concern

The literature regarding hydrogen sulfide toxicity indicates that, at high concentrations, hydrogen sulfide can affect the respiratory tract, eyes, and nervous system. Having limited exposure data in humans hinders the ability to precisely define the concentration at which any effects occur (called a threshold). Nausea, tremors, and breathing difficulties have been reported but only after extreme cases of acute poisoning. A review of the epidemiology evidence for chronic exposures indicates that respiratory symptoms (such as cough, wheezing, and shortness of breath) are the most common adverse effects reported at concentrations less than 10,000 parts per billion (ppb), and these effects appear to be temporary, as there are no accompanying effects on lung function. Exposure to >1,000 ppb hydrogen sulfide can also irritate the respiratory system in experimental animals. Eye irritation has been reported in workers following prolonged exposures to hydrogen sulfide concentrations as low as 700 ppb.

There are no toxicity studies of humans exposed to methyl mercaptan alone. Studies in experimental animals indicate no adverse effects of methyl mercaptan at repeated exposure concentrations of at least 57,000 ppb or acute exposure concentrations of at least 250,000 ppb.

## 1.2 Health Effects of Odors

The scientific literature demonstrates that odor itself is not an indicator of toxicity or adverse health effects. This is because many odorous substances have low odor thresholds (*i.e.*, the minimum concentrations at which people can smell specific odors) relative to levels that are of toxicological concern. As such, people can smell chemicals at concentrations that do not cause any health effects. Physical symptoms can occur in certain people in response to an odorous chemical at exposure concentrations below its toxicity threshold as a result of stress-induced physiological responses to perceptions of environmental risk. These symptoms

are not a result of a chemical-induced toxicological mechanism, so they do not indicate that the source of the odor caused a health effect.

### **1.3 Health Effects Claimed by Plaintiffs**

The Plaintiffs allege exposure to odors from the New-Indy paper mill caused or contributed to several health conditions. Most of the alleged conditions are prevalent in the general population and have many known risk factors. There are no reliable studies reporting associations between hydrogen sulfide or methyl mercaptan and any of the alleged health effects, except for eye and nasal irritation, shortness of breath, and headaches, but these effects only occur at very high hydrogen sulfide exposures, several times higher than those recorded by the community air monitors.

### **1.4 Risk Assessment**

The Plaintiffs' maximum estimated exposures to hydrogen sulfide were almost all below exposure guidance values and all were well below exposures associated with health effects in the literature, indicating that any exposures to hydrogen sulfide were not the cause of any of the Plaintiffs' claimed health effects.

### **1.5 Evaluation of Plaintiffs' Experts' Opinions**

Dr. Hecht, Dr. Barsotti, Dr. Palevsky, Dr. Fee, and Dr. Meggs all opined that hydrogen sulfide exposures associated with the New-Indy paper mill resulted in health conditions in nearby residents. None adequately considered the scientific evidence with respect to hydrogen sulfide and health effects. They did not provide any evidence that exposures associated with the New-Indy paper mill were sufficient to cause any health effects, much less those noted in Dr. Meggs's Independent Medical Exams (IMEs). None considered alternative causes for any claimed or reported condition or provided evidence that members of the larger population who were not evaluated likely had similar health conditions, including children or adolescents. None considered whether any health effects associated with odors were psychological *versus* toxic effects of odorous chemicals. Finally, Dr. Meggs's discussion of US EPA's reference concentration (RfC) was incorrect.

## 2 Credentials

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I am an epidemiologist and board-certified toxicologist with expertise in human health risk assessment. I am a fellow of both the American College of Epidemiology and the Academy of Toxicological Sciences (ATS), and I am currently on the Board of Directors of ATS. I am a Principal at Gradient, an environmental and risk sciences consulting firm. From 2009 to 2017, I was an adjunct faculty member in the Department of Epidemiology at the Harvard T.H. Chan School of Public Health. My *curriculum vitae* and testimony experience for the past 4 years are provided in Appendices A and B, respectively.

I received an S.B. degree in environmental engineering science from the Massachusetts Institute of Technology in 1996. I received an Sc.M. in epidemiology and a Ph.D. in environmental health sciences/toxicology from the Johns Hopkins Bloomberg School of Public Health in 2000 and 2002, respectively. From 2002 to 2004, I was a Cancer Prevention Fellow at the National Cancer Institute, where I conducted several molecular epidemiology studies on colon, breast, and prostate cancers, and was instrumental in the development of "Polymorphism Interaction Analysis," a statistical tool for cancer risk assessment. In 2004, I joined Gradient. My consulting practice consists of evaluating toxicity, epidemiology, and exposure data in the context of evaluating human health risks from substances in consumer products, pharmaceuticals, and medical devices, as well as from occupational and environmental exposures.

Based on my experience and expertise, I work with several organizations on issues relating to toxicology, epidemiology, risk assessment, and public health. Since 2008, I have served as an elected member of the Board of Health in Canton, Massachusetts, the community in which I reside. In this capacity, I provide advice on a broad range of public health topics, from the evaluation of chemical risks to the prevention of coronavirus disease 2019 (COVID-19). I am also a member of the Massachusetts Medical Reserve Corps and the Massachusetts Environmental Justice Assistance Network. In May 2012, I served as a panelist at a US EPA meeting that addressed how mode-of-action (MoA) evidence should be used in assessments of exposures to and health effects caused by chemical mixtures. In 2013, I served as an expert external peer reviewer for US EPA's "Provisional Peer-Reviewed Toxicity Values for Styrene-Acrylonitrile (SAN Trimer)" report. In 2014, I served as a reviewer for research grants submitted to the National Science Foundation, the California Breast Cancer Research Program, and the John Templeton Foundation. In 2016, I served as a reviewer for a K99 research grant submitted to the National Institute for Occupational Safety and Health (NIOSH). In 2017 and 2018, I served as a reviewer for several R21 research grants submitted to the National Institutes of Health.

I have been active in the Society of Toxicology for many years; I was previously the treasurer/secretary of the Risk Assessment Specialty Section and an elected member of the Nominating Committee. I am also active in the American College of Epidemiology, for which I served on the Board of Directors, and the Society for Risk Analysis. I taught "Research Synthesis and Meta-Analysis," a graduate-level course at the Harvard T.H. Chan School of Public Health. As reflected in my *curriculum vitae*, I have authored over 150 original peer-reviewed research articles, review articles (including systematic reviews, meta-analyses, and weight-of-evidence evaluations), and book chapters on a wide variety of chemicals and health outcomes in peer-reviewed journals, books, and meeting proceedings. I was on the editorial boards of *Carcinogenesis* and *The Open Biomarkers Journal* and was a managing editor of the *Journal of Environmental Protection Science*. I have been a peer reviewer for more than 35 journals.

## 3 Methodology

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My opinions are based on a review of a substantial number of documents, as well as my training and experience in toxicology, epidemiology, and risk assessment. The types of information on which I relied for this report include: case-specific documents; publicly available environmental and regulatory documents authored by agencies such as the US Agency for Toxic Substances and Disease Registry (ATSDR), US EPA, the California Office of Environmental Health Hazard Assessment (CalOEHHA), International Agency for Research on Cancer, and the World Health Organization; general scientific and regulatory literature in the fields of toxicology, epidemiology, and risk assessment; and scientific literature on hydrogen sulfide and methyl mercaptan.

The Plaintiffs discuss many health conditions that they allege are associated with hydrogen sulfide and methyl mercaptan. I assessed general causation by evaluating whether exposure to hydrogen sulfide or methyl mercaptan could cause or contribute to health conditions, and, if so, under what exposure conditions. In addition to relying on reputable secondary sources, including environmental and regulatory documents published by the agencies listed above, I also performed targeted literature searches using PubMed and Scopus. Below, I describe the main features of toxicity and epidemiology studies and discuss how I integrated evidence from these studies for my evaluation.

### 3.1 Epidemiology

Epidemiology is the study of the causes, distribution, and control of disease in populations (Greenland and Rothman, 2008). In epidemiology studies, both exposure and health outcomes are measured or estimated, and statistical analyses are conducted to assess whether there are associations between the two. These studies involve realistic exposure conditions and do not require extrapolation of results from animal studies. However, because these studies involve people living in the real world, study subjects often have other exposures or attributes that make it difficult to determine whether an association is causal, and each epidemiology study may have other limitations that can affect the interpretation of results.

### 3.2 Toxicology

Toxicology is the study of the potentially adverse health effects of biological, chemical, and physical agents on living organisms (Hayes and Kruger, 2014). It encompasses studies of humans, experimental animals, isolated cells, and isolated molecules, including MoA studies that assess how agents may cause observed effects. An understanding of toxicology is necessary for determining to how much of an agent one can be exposed, and under what conditions, without the likelihood of harm. In addition to an exposure assessment (*i.e.*, whether an individual had any contact with the agent in question and, if so, to what degree and under what conditions), a determination of dose (the amount of a chemical taken into the body over time) is a key component of evaluating health effects from biological, chemical, and physical agents. Factors that may influence the toxicity in individuals include genetic background, sex, age, health status, behavioral traits (*e.g.*, smoking and alcohol use), diet, and nutritional status (Aleksunes and Eaton, 2019).

Although virtually every agent is capable of producing toxic (or adverse) effects at some dose, the range of doses necessary to produce adverse effects, injury, or death varies widely among agents (Aleksunes and Eaton, 2019; Faustman, 2019). Both the nature and severity of effects from an agent vary with dose, and

the evaluation of the relationship between exposure to an agent and health effects is referred to as a dose-response assessment. The body has many biochemical and physiological processes that allow it to counteract an agent's adverse effects, and most agents do not cause adverse effects unless one is exposed to a dose sufficient to overwhelm the body's normal processes for a certain period of time. As such, many agents are not harmful when one is exposed to low doses (see, for example, Aleksunes and Eaton, 2019). In other words, there is a threshold dose below which there is no evidence of adverse health effects. Also, some agents provide beneficial effects at low doses.

The frequency and duration of exposure to an agent are also critical factors for determining toxicity; the adverse effects of an agent can also differ depending on whether exposure is to a single, large dose (acute exposure) or to lower doses over a long period of time (chronic exposure). For example, in the case of ethyl alcohol, a single large acute dose can cause severe adverse effects in the central nervous system, whereas chronic exposure to lower doses over a long period of time can damage the liver and cardiovascular system (Bruckner *et al.*, 2019). For most agents, one or a few small doses will either not result in any health effects or will result in only minor health effects; the severity of health effects is typically much greater for acute, large, single-dose exposures. With chronic exposure to sufficiently low doses, the body is able to eliminate the dose *via* excretion and repair any damage that may have occurred or adapt and find other means of accommodating the exposure (Aleksunes and Eaton, 2019).

For this report, I reviewed studies that evaluated the effects of inhalation exposures. I did not review studies that evaluated other routes of exposure (*e.g.*, dermal, oral, subcutaneous, intraperitoneal, or intravenous injection) because they are not relevant to human environmental exposures and are not representative of the exposures claimed by the Plaintiffs.

### 3.3 Evidence Evaluation

To determine whether, and under what conditions, exposure to hydrogen sulfide and methyl mercaptan could cause or contribute to health conditions, I evaluated relevant toxicity and epidemiology studies, as well as comprehensive reviews of these studies, to determine the overall plausibility for causality for health effects in humans, bearing in mind study quality and relevance, and uncertainties and inconsistencies in the evidence. I also considered reviews of these chemicals from regulatory and other authoritative bodies. Finally, I considered the exposure conditions under which any reported effects occurred so that I could determine whether they were relevant to the exposures claimed by the Plaintiffs.

### 3.4 Surveys

Surveys and questionnaires can gather detailed information regarding a wide range of health effects, exposures, and other potential risk factors from a group of respondents (Aschengrau and Seage, 2003). They can be conducted by face-to-face or telephone interviews or they can be self-administered. However, the study design, interviewer, and respondent can all introduce survey errors (Aschengrau and Seage, 2003).

For any type of survey, higher participation rates lead to more accurate results. A low participation rate can skew results, as it cannot be known how non-participants (including those not contacted and those who are contacted but refuse to participate) would have answered the survey, or whether those who did not participate are similar to those who did. This is called selection bias, which occurs when there is an error in choosing the individuals or groups to take part in a study. If there are important differences between participants and the larger population from which they are drawn, the results of the study may not be valid. Self-selection can also skew outcomes when people with relevant characteristics differ in their propensity to come forward (Rothman, 2002; Aschengrau and Seage, 2003).

Each survey method has strengths and limitations. Personal interviews are generally the most labor- and cost-intensive of the three methods but allow for the incorporation of visual aids and clarification (decreasing the potential for question misinterpretation). Also, the greater intimacy may increase the subject's willingness to participate. On the other hand, personal interviews are prone to interviewer error, in which the interviewer does not follow the same protocol for all subjects or prompts subjects to answer in a specific way (Armstrong *et al.*, 1994). Typically, personal interviews increase the length of individual responses, but the effect of the interviewer on the accuracy of responses is unknown.

Telephone interviews elicit shorter responses than personal interviews and may lead to less accurate information. For example, participants are likely to choose from the first or last answer when given a list over the phone (Armstrong *et al.*, 1994).

Self-administered questionnaires are the most cost-effective but least easily monitored method. Self-administered questionnaires may yield more accurate data on sensitive or embarrassing topics and allow more time for subjects to reflect on the questions and recall relevant details, but they are also subject to misunderstood questions, lower response rates, and higher incompleteness rates (Aschengrau and Seage, 2003; Armstrong *et al.*, 1994). The most effective self-administered questionnaires are short, do not contain open-ended questions or questions that require probes or complex branching (*e.g.*, if you answered yes, go to question 4; if you answered no, go to question 6), and do not require questions to be answered in a strict order (Armstrong *et al.*, 1994).

The wording and length of the survey can affect the quality of data collected. For example, simple words like *anyone*, *most*, and *average* can be interpreted in many ways (Groves, 1989, as cited in Greenland and Rothman, 2008). If medical terms or jargon are not explained in simple terms, this could lead to skipped or misinterpreted questions. Shorter questions, with fewer ideas in them, are generally clearer. Longer surveys increase the risk of break-offs or discontinuance.

Surveys collect subjective data reliant on human knowledge and memory, which are prone to errors due to memory limitations and recall bias. In general, an increase in time between exposure and the survey leads to greater memory limitations of specific details (Armstrong *et al.*, 1994; Aschengrau and Seage, 2003). Respondents' recall or reporting may also be subjected to their surrounding circumstances. For example, respondents with particular health effects are more likely to report past exposures, and respondents who believe they were exposed to a substance are more likely to report symptoms or illnesses. Plaintiffs in a lawsuit also may be more likely to remember exposures and health effects (Lees-Haley *et al.*, 1996, 1997). In addition, over-reporting may occur when recall is requested for a particular period (Armstrong *et al.*, 1994). This form of recall bias, referred to as telescoping, occurs when subjects report exposures that occurred outside the exposure period as occurring during the exposure period.

Respondents may also be influenced by social desirability bias, in which they under-report socially undesirable behaviors (Armstrong *et al.*, 1994). It has also been reported that surveyed subjects will often report what they believe the investigator expects or report what reflects positively on their own abilities, knowledge, beliefs, or opinions (Cook and Campbell, 1979).

Similar issues arise with self-reported health data, particularly when they are not confirmed by medical personnel. While self-reported data are generally valid for diseases that have distinct diagnostic criteria (*e.g.*, bone fractures, hypertension, cancers of the bowel or breast), they are often not valid for health effects with less clear diagnostic criteria. For example, an evaluation of self-reported data in the Nurses' Health Study found a much lower level of confirmation between self-reported data and confirmed medical records for lung cancer, myocardial infarction, and stroke, all of which have less clear diagnostic criteria (Colditz *et al.*, 1986, as reported in Hennekens *et al.*, 1987). Similarly, when Zhu *et al.* (1999) compared self-

reported data with medical record information in a prostate cancer case-control study, they found that the two data sources had very similar demographic and anthropometric information, substantially similar data regarding the history of inguinal hernia and kidney stones, and moderately similar data on vasectomy, family history of prostate cancer, smoking, and alcohol consumption. The two data sources were poorly concordant for prior diseases with less explicit diagnostic criteria (*i.e.*, benign prostatic hyperplasia and prostatitis) (Zhu *et al.*, 1999).

The most robust surveys address the above limitations by validating at least a subset of responses. For example, exposure estimates can be confirmed by measurements, health outcomes can be validated by physicians, and characteristics of non-participants can be ascertained to determine how they differ from participants. These "reliability checks" can be informative regarding the quality of survey responses.

### 3.5 Exposure Assessment

An exposure assessment (*i.e.*, an assessment of whether an individual had any contact with the chemical/substance in question and, if so, to what degree) and a determination of dose (the amount of a chemical/substance taken into the body over time) are key components for evaluating the potential for adverse health effects from exposure to chemicals and other substances. A number of factors affect the total amount of a substance to which a person is exposed and how much of this substance is then able to enter the body and interact with tissues and organs. Thus, a key element of an exposure assessment is to characterize the specific exposure scenario for the population or person being studied. This involves determining how a chemical or substance moves from the source, through the environment, and to the population or person being exposed (the exposure pathway); the means of entry into the body (*i.e.*, *via* inhalation, ingestion, or skin contact, known as the exposure route); and the intensity (*e.g.*, concentration), frequency (*e.g.*, how many times a person is exposed per day), and duration (*e.g.*, how many weeks or years total) of exposure.

Even a highly toxic chemical or substance cannot cause health effects in individuals in the absence of exposure. Conversely, if the received dose is extremely high, otherwise innocuous or beneficial substances can cause harm. For example, water intake is essential for humans, but drinking too much can lead to water intoxication, severe disruption of ion concentrations, and a potentially fatal outcome (Farrell and Bower, 2003).

Exposure can be estimated based on many types of information, including direct measurement of chemicals or substances in environmental media (*e.g.*, air, soil, or dust); measurements of substances or metabolites in biological samples, such as blood and urine (*i.e.*, exposure biomarkers); and through exposure modeling. More in-depth exposure assessments may also examine specific internal exposure targets (*e.g.*, a specific target organ) or estimate an internal dose (*i.e.*, the amount of the substance that reaches a specific target tissue). Depending on the types of information available, exposure assessments can yield quantitative, semi-quantitative, or qualitative exposure estimates.

In this case, I relied on air monitoring data collected in the community and at the fenceline of the New-Indy paper mill to conduct an exposure assessment.

### 3.6 Exposure Guidance Values

Exposure guidance values are health-based guidelines or regulatory limits that are generally equivalent to a daily exposure to a substance not associated with a risk of health effects. If the concentration of a substance in environmental media does not exceed an exposure guidance value, then it can be concluded

that the exposure does not present a toxicological concern and there is no need for additional analysis. Exceedance of an exposure guidance value means that further analysis may be required, but does not indicate that a health effect will occur. As stated by retired US EPA toxicologist Dr. John Lipscomb (2019):

The federal government has the responsibility to protect human health against the harmful effects of chemical exposure, and uses rather conservative policies and procedures to develop regulatory standards. These protective risk values typically do not inform the likelihood of harm or the type of harm that should be anticipated if regulated exposure standards are exceeded.

For many substances, there is a threshold dose below which adverse health effects do not occur, and toxicity or epidemiology studies can be used to determine a lowest observed adverse effect level (LOAEL) and a no observed adverse effect level (NOAEL) for oral or dermal exposures, or a no observed adverse effect concentration (NOAEC) and lowest observed adverse effect concentration (LOAEC) for inhalation exposures. The NOAEL (or NOAEC) is the highest dose (or exposure) to which a human or other animal is exposed in a study without experiencing any adverse health effects. It is assumed that any dose/exposure below the NOAEL/NOAEC can be safely tolerated for a period of time corresponding to the exposure duration of the study in which it was identified. The LOAEL/LOAEC is the lowest dose/exposure that results in an adverse health effect in a study.

To develop exposure guidance values, government agencies and other organizations select a NOAEL/NOAEC (or, if a NOAEL/NOAEC is not identified, a LOAEL/LOAEC) for the most sensitive endpoint (usually the adverse effect that occurs at the lowest dose/exposure level) in the most sensitive species. This NOAEL/NOAEC or LOAEL/LOAEC is called the "critical effect level" and is selected after a careful review of all relevant toxicity and epidemiology studies. Once the critical effect level is identified, it is divided by uncertainty factors (UFs) to provide additional protection.<sup>1</sup> Because exposure guidance values are derived based on the lowest dose or exposure associated with harm in the most sensitive species, these values should protect even the most sensitive individuals from all potential effects of the chemical.

For chronic effects, the most common exposure guidance values used are the reference dose (RfD), for substances that are ingested or absorbed through the skin, and the RfC, for inhaled substances. The RfD and RfC are estimates of a daily oral exposure or continuous inhalation exposure, respectively, that are likely to be without an appreciable risk of deleterious effects during a lifetime (US EPA, 2011). However, adverse health effects will not necessarily occur even at exposures greater than the RfD or RfC, or greater than other any other exposure guidance value, for that matter.

The RfD and RfC were developed by the US EPA National Center for Environmental Assessment for use in risk assessments by US EPA, state and local health agencies, other US federal agencies, and international organizations (US EPA, 2019). They are applicable to the assessment of risks in the general population. As stated by Dr. Lipscomb (2019):

While RfD values are risk values, their conservative biases and their intended health protective application preclude their ability to quantify risk. RfD values are developed very specifically for the purpose of protecting humans against the potential harm from what might be a worst-case exposure (chronic, lifetime) scenario. Said another way, the US EPA's RfD values protect against harm, when exposures can be controlled. Exposure

<sup>1</sup> A UF is one of several, often 10-fold, default factors that are intended to account for: (1) variation in susceptibility among the members of the human population (*i.e.*, interindividual or intraspecies variability); (2) uncertainty in extrapolating from animal data to humans (*i.e.*, interspecies uncertainty); (3) uncertainty in extrapolating from data obtained in a study with less-than-lifetime exposure (*i.e.*, extrapolating from subchronic to chronic exposure); (4) uncertainty in extrapolating from a LOAEL rather than from a NOAEL; and (5) uncertainty associated with extrapolation when the database is incomplete (US EPA, 2011).

limits...are regulatory, enforceable standards that have their quantitative basis in RfD values, which in turn representing exposures anticipated an absence of chemical induced adverse effects can be anticipated with confidence.

There are additional exposure guidance values for workers in occupational settings, which are called occupational exposure limits (OELs). OELs are regulations, guidelines, or recommendations established by several agencies, and they include Occupational Safety and Health Administration (OSHA) permissible exposure limits, American Conference of Governmental and Industrial Hygienists threshold limit values, and NIOSH recommended exposure limits. Although the specific definitions for each vary, OELs are generally intended to represent time-weighted average concentrations of chemicals in air during an 8-hour work day to which nearly all workers can be exposed over a working lifetime without adverse health effects.

There are substantial differences in how toxicity data are used in a regulatory framework to derive exposure guidance values to protect public health *versus* for calculating specific risks (Aleksunes and Eaton, 2019). This is because regulatory toxicologists do not estimate the likelihood of health effects actually occurring in a population or an individual (US EPA, 2004a; ATSDR, 2018a). Rather, regulators use high-end estimates of exposure and toxicity (that generally result in overprediction of potential health risks) to be protective of human health. The aim of US EPA and other public health agencies is not to precisely define which effects are expected to occur, but to define the level at which health effects are unlikely to occur (US EPA, 1993; ATSDR, 2013). US EPA's guidelines for regulatory criteria development state that such criteria are applicable to "susceptible groups," or sensitive subpopulations, which include life stages and other factors that may predispose individuals to have a greater response to an exposure (US EPA, 2002a; CalOEHHA, 2008a).

Below, I discuss exposure guidance values for hydrogen sulfide and methyl mercaptan. Because only hydrogen sulfide data were available to me, I performed a risk assessment based on the concentrations to hydrogen sulfide that have been associated with health conditions.

### **3.7 Risk Assessment**

To assess whether the Plaintiffs could be at risk for any health conditions as a result of exposure to hydrogen sulfide, I compared their potential exposures based on air monitoring data to concentrations that have been associated with their claimed health conditions.

### **3.8 Evaluation of Plaintiffs' Expert Reports**

I reviewed the opinions of Plaintiffs' experts, Dr Hecht, Dr. Barsotti, Dr. Palevsky, Dr. Fee, and Dr. Meggs, with a particular focus on their methodologies. I also assessed whether their conclusions are supported by the scientific evidence.

## 4 Health Conditions Claimed by the Plaintiffs

In their deposition testimonies, the Plaintiffs claim to have developed various health effects from exposures to odorous gases originating from the New-Indy paper mill, as summarized in Table 4.1. Specifically, the Plaintiffs claim that they were exposed to hydrogen sulfide and methyl mercaptan, and that these exposures occurred at their properties in the community near the paper mill. I evaluated whether it can be concluded to a reasonable degree of scientific certainty that Plaintiffs' alleged health effects were attributable to exposures to odorous substances (specifically, hydrogen sulfide and methyl mercaptan) that allegedly emanated from the New-Indy paper mill.

**Table 4.1 Health Effects Claimed by Plaintiffs**

Plaintiff Name	Claimed Health Conditions		Source
Candice Cherrybone	Burning eyes Runny nose	Headaches	Cherrybone, 2023
Melda Gain	Headaches Nosebleeds Insomnia/inability to sleep Sinus problems	Nausea Dizziness Anxiety	Gain, 2023a
Orrin Gain	Headaches Difficulty breathing/ shortness of breath	Coughing Insomnia Anxiety	Gain, 2023b
Marty Kennedy	Sleep problems Stress/anxiety (contributing to atrial fibrillation) Nausea/sour stomach Nasal irritation Nosebleeds Fatigue	Thumping in ears Headaches Coughing Sore throat Sinus problems Dizziness/equilibrium issues	Kennedy, 2023a,b
Terri Kennedy	Bronchitis Allergic reactions/hives/ rashes Sleep problems Headaches	Fatigue Mental distress/anxiety Vertigo/balance issues Nausea Sinus problems	Kennedy, 2023c,d
Enrique Lizano	Headaches Vertigo/balance issues Ears ringing	Body tremors Anxiety/stress Sleep problems	Lizano, 2023a,b
Sansanee Lizano	Migraines/headaches Nausea (due to migraine)	Anxiety/stress	Lizano, 2023c
Michael (Shane) Nickell	Migraines/headaches Itchy eyes	Throat congestion	Nickell, 2023a
Tracie Nickell	Severe migraines Lightheadedness Memory issues Dry eyes	Cough Shortness of breath Nausea Sleep problems	Nickell, 2023b

Plaintiff Name	Claimed Health Conditions		Source
Amanda Swager	Sinus pain Burning/sore eyes Headaches Nausea/loss of appetite Cough Fatigue	Sore throat Sleep problems Anxiety Nosebleeds Dizziness	Swager, 2023a, b
Shara Swager	Headache Sore throat Nausea	Sleep problems Anxiety Dizziness	Swager, 2023c,d
Kenny White	Eye irritation Sinus problems	Headaches Nasal irritation	White, 2023

Below, I describe the prevalence of each claimed condition in the US, which indicates how common each is in the general population; I then summarize risk factors for these conditions. Overall, in my opinion, none of the Plaintiffs was exposed to concentrations of hydrogen sulfide or methyl mercaptan that could have caused or contributed to their claimed health conditions.

## 4.1 Prevalence

Public health agencies and researchers often track the prevalence of health conditions in the general population. US prevalence data for the health conditions claimed by the Plaintiffs are shown in Table 4.2. The Plaintiffs' claimed conditions are fairly common. The reported prevalence of these health conditions may be underestimated because they are underreported (*e.g.*, people with headaches or a cough may not go to the doctor).

**Table 4.2 Prevalence of Claimed Health Conditions in the US**

Health Condition	Frequency	Notes	References
Anxiety	31.1%	Prevalence of any anxiety disorder during lifetime among US adults	NIMH (2017)
Balance problems	20.6%	Adults in the US reporting vestibular problems in a 12-month period	ASHA (2021)
Breathing difficulty (dyspnea)/shortness of breath/wheezing	25%	Percentage of outpatients	Berliner <i>et al.</i> (2016)
Congestion (nasal)	11.5%	Demographically and geographically representative sample of 10,336 US adults	Palmer <i>et al.</i> (2019)
Conjunctivitis/eye irritation	8.1%	Percentage of US residents	McCann <i>et al.</i> (2022)
Cough	5-40%	Prevalence depends on smoking status	Cleveland Clinic (2014)
Dizziness/faintness	30%	Study of adults $\geq 65$ years old who experience some form of dizziness	ASHA (2017a)
Fatigue/lethargy	Males: 10.1% Females: 15.3%	Prevalence in general population	CDC (2013)
Headache	Males: 10.6% Females: 20.1%	Defined as migraine or severe headaches	CDC (2020)

Health Condition	Frequency	Notes	References
Insomnia/difficulty sleeping	35.2%	Percentage of adults who reported short sleep duration (less than 7 hours of sleep per 24-hour period)	CDC (2017)
Nausea and vomiting	9.5%	Study of gastrointestinal symptoms in the past week in nationally representative survey of 71,812 people in the US	Almario <i>et al.</i> (2018)
Neuromuscular issues (weakness, numbness, muscle pain, tingling, motor coordination issues)	25-30%	Estimated percentage of Americans affected by muscular neuropathy	Cleveland Clinic (2019a)
Nosebleeds	60%	Percentage of people who will have a nosebleed during their lifetime	Tabassom and Cho (2017)
Rash/skin irritation	10.2%	Percentage of adults (18 and over) with dermatitis or any other red, inflamed skin rash	CDC (c. 2014)
Rhinosinusitis/sinus pain	11.6%	Age-adjusted prevalence for adults	CDC (2018a)
ringing in ears/ tinnitus	15-20%	Estimated percentage of adults affected	Mayo Clinic (2021a)
Sore throat	2-4%	Based on family physician visits	Worrall (2011)
Subjective cognitive decline (cognitive/ memory/confusion/ communication issues)	11.1%	US adults who reported experiencing subjective cognitive decline (SCD)	CDC (2018b)
	11.7%	US adults $\geq 65$ years old who reported experiencing SCD	
	10.8%	US adults 45-64 years old who reported experiencing SCD	

Notes:

ASHA = American Speech-Language-Hearing Association; CDC = Centers for Disease Control and Prevention; NIMH = National Institute of Mental Health; SCD = Subjective Cognitive Decline; US = United States.

## 4.2 Risk Factors

Each of the specific health conditions mentioned by the Plaintiffs in their depositions has several known risk factors. It is possible that some people with one or more risk factors for a health condition never develop that condition, and it is also possible that a person with a health condition has few or no known risk factors.

In Table 4.3, I summarize the risk factors I identified for the specific health conditions that the Plaintiffs claim, as determined by medical and public-health organizations (*e.g.*, Mayo Clinic, National Institutes of Health), and as presented in the published scientific literature. The established risk factors for each condition in Table 4.3 have been studied extensively, and the evidence to support them is generally strong and consistent.

**Table 4.3 Risk Factors for Claimed Health Conditions**

Health Condition	Risk Factors	References
<b>Anxiety</b>		
Anxiety describes a group of disorders that manifest recurring intrusive thoughts and concerns. Symptoms of these disorders include sweating, trembling, dizziness, and rapid heartbeat.	Temperamental traits ( <i>e.g.</i> , shyness, behavioral inhibition in childhood) Experiencing or witnessing trauma Stress Personal or family history of anxiety or other mental illness Health conditions ( <i>e.g.</i> , thyroid problems, heart arrhythmias) Caffeine or substance/medication use	American Psychological Association (2021); NIMH (2018); Mayo Clinic (2018a)
<b>Balance Problems</b>		
The human balance system incorporates input from the vestibular, visual, and somatosensory systems. A balance system disorder involves disturbance of one or more of these systems. Symptoms include dizziness, lightheadedness, and vertigo.	Acute injury to the vestibular system Effects of age on the vestibular system Use of alcohol or drugs Anatomic brain changes Inner ear problems ( <i>e.g.</i> , inflammatory process, autoimmune disease, Ménière's disease) Benign paroxysmal positional vertigo Circulatory or cardiovascular conditions Cochlear implant surgery Enlarged vestibular aqueduct syndrome Genetic disorders Viral or bacterial infections Medication side effects Metabolic disorders Musculoskeletal conditions Neurological impairments Otosclerosis Ototoxicity Peripheral neuropathy Psychological disorders Lesions on the auditory nerve Temporal bone fracture Traumatic brain injury Vestibular migraine or other migraine variants	ASHA (2021)
<b>Congestion (Nasal)</b>		
Congestion occurs when respiratory tissues and blood vessels become swollen with excess fluid. This may or may not cause mucus discharge. Symptoms usually include chronic sneezing, a drippy nose, and other upper respiratory reactions.	Exposure to irritants, including tobacco smoke Allergies Age (older than 20) Prolonged use of decongestant drops or sprays Sex (female) Occupational exposure to fumes Chronic health conditions ( <i>e.g.</i> , hypothyroidism, chronic fatigue syndrome, diabetes)	Mayo Clinic (2021b); Cleveland Clinic (2019b)

Health Condition	Risk Factors	References
<b>Conjunctivitis/Eye Irritation</b>		
Irritation of the eyes can be felt as pain or itchiness of the eyes or eyelids.	Seasonal and/or environmental allergies Exposure to irritants (e.g., tobacco smoke, chlorinated pool water) Contact lenses Eye infections or blepharitis Dry eye Other medical conditions (e.g., Sjogren's syndrome, rheumatoid arthritis)	Ramsey <i>et al.</i> (2017); WebMD (2016); NEI (2017)
<b>Cough</b>		
Coughing is a symptom that can be classified based on how long it lasts and by other features. Chronic cough refers to a cough that lasts for more than 8 weeks.	Smoking Allergies Exposure to environmental irritants Chronic respiratory conditions (e.g., asthma, COPD, tuberculosis, lung cancer) Heart failure Postnasal drip GERD	American Lung Association (2016)
<b>Dizziness</b>		
The term "dizziness" is nonspecific and can refer to symptoms such as lightheadedness or vertigo.	Viral or bacterial infections Acute injury to the head (e.g., traumatic brain injury) or vestibular system Disorders of the vestibular or balance systems (e.g., Meniere's disease, otosclerosis, enlarged vestibular aqueduct syndrome) Neurological conditions (e.g., Parkinson's disease, multiple sclerosis) Blood pressure changes Vascular problems Visual disorders Anxiety disorders Low iron levels Hypoglycemia Overheating and dehydration Medication side effects Alcohol use Heart failure	ASHA (2017b); Mayo Clinic (2020a); American Addiction Centers (2022); ESC HFA (2022)

Health Condition	Risk Factors	References
<b>Fatigue/Lethargy</b>		
Fatigue can be manifested as difficulty initiating activity, reduced capacity maintaining activity, difficulty with concentration, or emotional instability.	Lifestyle factors ( <i>e.g.</i> , lack of exercise, unhealthy eating habits, and use of alcohol and drugs) Psychiatric disorders ( <i>e.g.</i> , depression) Infections ( <i>e.g.</i> , mononucleosis) Metabolic disorders Autoimmune diseases Sleep disorders Nutritional deficiencies Cancer or diseases of other organ systems ( <i>e.g.</i> , diabetes, COPD, coronary artery disease) Medication side effects Congestive heart failure	A.D.A.M. Medical Encyclopedia (2015); Mayo Clinic (2016a, 2020, 2021)
<b>Headache</b>		
Headache is pain in any region of the head. A headache may appear as a sharp pain, a throbbing sensation, or a dull ache. Headaches can develop gradually or suddenly and may last from less than an hour to several days.	Sex (female) Alcohol consumption Anxiety or depression Sleep disturbances Obesity Smoking Dehydration Head or neck injury Hypertension Rhinosinusitis Brain arteriovenous malformations Head trauma/concussion Overuse of caffeine Cocaine use Overuse of headache medication Other chronic pain conditions	Mayo Clinic (2018a2018b, 2018c3485; 2022); Scher <i>et al.</i> (2008); Arca and Halker Singh (2021); MedlinePlus and NLM (2020); National Headache Foundation (2007)
<b>Insomnia/Difficulty Sleeping</b>		
Insomnia is a sleep disorder defined by chronic difficulty with sleep initiation, duration, consolidation, or quality despite ample time allotted for sleep.	Age (older adults) Sex (females) Medical and mental health issues ( <i>e.g.</i> , pain, anxiety, depression) Stressful life events Hereditary conditions Lifestyle ( <i>e.g.</i> , smoking, exercise, sleep habits) Cocaine use	Bhaskar <i>et al.</i> (2016); Leblanc <i>et al.</i> (2009); MedlinePlus and NLM (2020)

Health Condition	Risk Factors	References
<b>Nausea and Vomiting</b>		
Nausea is an uneasiness of the stomach that accompanies the urge to vomit. Vomiting describes the forcible emptying of the stomach contents through the mouth. Nausea and vomiting are symptoms of numerous health conditions.	Pregnancy, particularly in the first trimester Cancer treatment Food poisoning Motion sickness Intense pain Emotional stress or fear Smells or odors Other medical conditions ( <i>e.g.</i> , gallbladder disease, viral infections, concussions, appendicitis, migraines) Exposure to certain chemicals	Cleveland Clinic (2019c)
<b>Neuromuscular Issues</b>		
Neuromuscular issues generally affect the nerves that control voluntary movement and relay sensory information to the brain. This can lead to neurons becoming unhealthy or dying, negatively impacting muscular health and potentially leading to muscle atrophy. Symptoms include muscle weakness, twitching, cramps, aches, and pains; muscle loss; movement issues; balance problems; numbness, tingling, or painful sensations; droopy eyelids; double vision; trouble swallowing; and trouble breathing.	Older age Diabetes Heavy alcohol use Trauma and nerve compression ( <i>e.g.</i> , caused by repetitive motion or falls, accidents, fractures, and sports activities) Metabolic syndrome ( <i>i.e.</i> , high blood pressure, high cholesterol, obesity, and diabetes) Family history of neuromuscular disorders Spontaneous gene mutation Immune system disorders Certain infections ( <i>e.g.</i> , chickenpox, shingles, HIV, herpes, syphilis, Lyme disease, leprosy, West Nile Virus, hepatitis C, Epstein-Barr virus)	Cedars-Sinai (2021); Cleveland Clinic (2019a)
<b>Nosebleeds</b>		
Nosebleeds can occur when the lining of the nose, which contains many blood vessels that are close to the surface, is damaged.	Exposure to dry air Acute and chronic sinusitis Allergies Use of certain medications ( <i>e.g.</i> , aspirin, inhaled corticosteroids, blood thinners, nasal sprays) Bleeding disorders ( <i>e.g.</i> , hemophilia) Exposure to chemical irritants ( <i>e.g.</i> , ammonia) Cocaine use Common cold Deviated septum Foreign body in the nose and nasal trauma ( <i>e.g.</i> , nose-picking) Nonallergic rhinitis	Tabassom and Cho (2017); Mayo Clinic (2018d)

Health Condition	Risk Factors	References
<b>Rash</b>		
A rash occurs when a patch of skin becomes irritated or inflamed, resulting in changes in texture or color. The patch may become red, warm, scaly, bumpy, dry, itchy, swollen, blistered, cracked, or painful. A rash may occur all over the body or may be localized.	Age (infancy) Allergies and asthma ( <i>e.g.</i> , drug reaction, food or animal allergy) Occupation Health conditions ( <i>e.g.</i> , lupus, psoriasis, Lyme disease) Prolonged exposure to heat or irritants Insect bites ( <i>e.g.</i> , spider, mosquito, tick, flea) Infection Immune system disorders	NCI (c. 2021); Mayo Clinic (2019a)
<b>Ringling in Ears (Tinnitus)</b>		
Ringling in the ears, or tinnitus, is not caused by an external sound.	Exposure to loud noises Age (older) Sex (male) Tobacco and alcohol use Obesity Cardiovascular problems ( <i>e.g.</i> , high blood pressure) Arthritis Head injury Ear problems ( <i>e.g.</i> , Meniere's disease)	Mayo Clinic (2021a)
<b>Rhinosinusitis</b>		
Rhinosinusitis describes a common condition in which the sinuses, or cavities around the nasal passages, become inflamed and swollen.	Nasal passage abnormalities ( <i>e.g.</i> , deviated septum, nasal polyps) Asthma Aspirin sensitivity Immune system disorders ( <i>e.g.</i> , HIV, cystic fibrosis) Respiratory tract infections Allergies Regular exposure to cigarette smoke	Mayo Clinic (2016b)
<b>Shortness of Breath</b>		
Shortness of breath is characterized as discomfort and difficulty breathing, and the symptoms depend in part on the cause of the condition. Shortness of breath can be acute or chronic.	Respiratory conditions ( <i>e.g.</i> , asthma, pneumonia, COPD, restrictive lung disorders, pneumothorax, lung cancer, pleural effusion, pleurisy, pulmonary edema, COVID-19) Heart conditions ( <i>e.g.</i> , arrhythmias, heart attack, heart failure) Smoking and exposure to cigarette smoke Physical deconditioning Pulmonary embolism Anemia Metabolic acidosis Anxiety disorder ( <i>e.g.</i> , hyperventilation syndrome) Obesity Smoking and exposure to crack cocaine smoke Acid reflux and GERD	Merck Sharp & Dohme Corp. (2016); Mayo Clinic (2020b, 2020c, 2021c); MedlinePlus and NLM (2020); Zergham and Heller (2022)

Health Condition	Risk Factors	References
<b>Sore Throat</b>		
A sore throat is pain, scratchiness, or irritation that often worsens when swallowing.	Younger age Viral infections ( <i>e.g.</i> , common cold, flu, mononucleosis, measles, chickenpox, COVID-19, croup) Bacterial infections ( <i>e.g.</i> , strep throat) Exposure to tobacco smoke or use of tobacco products Allergies Alcohol use Medical conditions ( <i>e.g.</i> , sinus infections; GERD; tonsillitis; tumors of the throat, tongue, or larynx) Weakened immunity	Mayo Clinic (2020d, 2021d)
<b>Subjective Cognitive Effects</b>		
Mild cognitive impairment is the intermediate stage between expected cognitive decline from normal aging and more serious issues related to dementia.	Older age Family history High blood pressure/hypertension High cholesterol levels Diabetes Obesity Prior stroke Specific form of APOE e4 gene Hormone changes ( <i>e.g.</i> , pregnancy, menopause) Certain medical conditions ( <i>e.g.</i> , multiple sclerosis, chronic fatigue syndrome, lupus) Chemotherapy Cancer that affects the brain Depression Sleep problems ( <i>e.g.</i> , apnea, insomnia, narcolepsy) Smoking	Mayo Clinic (2020e); Cleveland Clinic (2019b); WebMD, LLC (2021)

## Notes:

A.D.A.M. = Animated Dissection of Anatomy for Medicine; ASHA = American Speech-Language-Hearing Association; CDC = Centers for Disease Control and Prevention; COPD = Chronic Obstructive Pulmonary Disorder; ESC HFA = European Society of Cardiology, Heart Failure Association; GERD = Gastroesophageal Reflux Disease; HIV = Human Immunodeficiency Virus; NCI = National Cancer Institute; NEI = National Eye Institute; NIMH = National Institute of Mental Health; NLM = National Library of Medicine.

## 5 Health Effects Associated with Odors

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Many chemicals have detectable odors that indicate their presence in air, but the detection of an odor is not an indicator of chemical exposures that are of toxicological concern. This is because the levels at which odors are detected (*i.e.*, odor detection thresholds) differ from the levels at which odorous chemicals may induce toxicity (*i.e.*, toxicity thresholds). To establish whether a health risk exists, the odorous chemical must be identified and its hazards understood. Strong odorants are not necessarily toxicants, and toxicants do not always produce an odor.

Odor detection and odor recognition thresholds are typically substantially lower than the nuisance threshold (Amoore and Hautala, 1983; Batterman *et al.*, 2023). The odor detection threshold "is the lowest concentration of odorant that will elicit an olfactory response without reference to odor quality in a specified percentage of a given population" (US EPA, 1992). The recognition threshold is "the minimum concentration that is recognized as having a characteristic odor quality by a specific percentage (usually 50%) of the population" (US EPA, 1992). That is, at lower concentrations people can detect an odor, but cannot identify what they are smelling until it reaches a higher concentration (US EPA, 1992). The nuisance threshold is the concentration at which an odor is perceived to be annoying (Batterman *et al.*, 2023).

Many airborne chemicals have established odor and irritation thresholds and, for these chemicals, olfaction occurs at lower levels than irritation, thus providing an early warning against potential irritation (Cometto-Muñiz and Cain, 1995, as cited in Dalton, 2003). For some chemicals that are strong odorants but weak irritants, such as hydrogen sulfide, the odor threshold concentration is several orders of magnitude lower (*e.g.*, hundreds or thousands of times less) than that for irritation (Shusterman, 2001). Put another way, someone can smell these chemicals at concentrations that are far too low to cause any irritation. Several compilations of odor and irritation thresholds for airborne chemicals reported no relationship between odor detectability and irritation (Amoore and Hautala, 1983; Ruth, 1986; Cometto-Muñiz and Abraham, 2016). In addition, toxicity thresholds for many odorous chemicals are much higher than their odor thresholds (*e.g.*, up to five orders of magnitude higher for hydrogen sulfide and methyl mercaptan, as discussed in Section 6 and 7), indicating that the potential toxicity of a chemical is not associated with its odorant properties.

Finally, the thought of an odor (or the sense of alarm from an odor) can cause effects such as increased heart rate, more forceful contractions or heart "pounding," sweating, nausea, headache, dizziness, anxiety, and panic, but it is not the source of the odor itself causing the effect (Smeets and Dalton, 2005; Shusterman, 1992). These effects cease once the odor is gone and are not a result of any tissue damage (Smeets and Dalton, 2005). Because most odorous chemicals typically have odor thresholds far lower than their toxicity thresholds, these symptoms are likely the result of non-toxicological mechanisms rather than true toxic effects (Bulsing *et al.*, 2009; Dalton, 1996, 2003; Devriese *et al.*, 2004; Neutra *et al.*, 1991; Schiffman and Williams, 2005; Shusterman *et al.*, 1991; Shusterman, 1992, 2001; Smeets and Dalton, 2005; Vrijheid, 2000).

## 6 Hydrogen Sulfide

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Hydrogen sulfide is a highly odorous gas. Natural sources include sulfur springs, swamps, undersea vents, stagnant bodies of water, and volcanoes (ATSDR, 2016; EC SCOEL, 2007). Hydrogen sulfide is also produced by bacteria during the decay of both plants and animal protein or through the direct reduction of sulfate, and it is a constituent of crude petroleum and natural gas (ATSDR, 2016; EC SCOEL, 2007). Industrial sources of hydrogen sulfide include municipal sewers, sewage-treatment plants, natural gas refineries, viscose rayon manufacturing, iron smelters, pulp and paper operations, farms, and landfills (ATSDR, 2016; EC SCOEL, 2007).

Hydrogen sulfide is also produced endogenously in the human intestinal tract and the mouth (ATSDR, 2016). As a component of bad breath (*i.e.*, halitosis), concentrations of 1-100 ppb have been measured in air from the mouth (ATSDR, 2016). In the intestinal tract, hydrogen sulfide can comprise up to 10% of intestinal gases, and in flatus, hydrogen sulfide concentrations of up to 18,000 ppb have been measured among individuals on a normal diet (ATSDR, 2016; Batterman *et al.*, 2023). Hydrogen sulfide is also used in a number of physiological functions in the human body, including in the cardiovascular system and in the brain (ATSDR, 2016).

Air concentrations of hydrogen sulfide from natural sources, such as swamps, bogs, and volcanoes, range from 0.11 to 0.33 ppb (ATSDR, 2016). In urban areas, air concentrations are generally less than 1 ppb, whereas concentrations in communities near natural sources of hydrogen sulfide or near industries releasing hydrogen sulfide are higher, often exceeding 90 ppb (ATSDR, 2016).

As discussed in Section 5, the odor detection and odor recognition thresholds can range considerably. According to Batterman *et al.* (2023), "inter-individual variation in sensitivity to H<sub>2</sub>S [hydrogen sulfide] is large," with average odor detection thresholds ranging from 0.07 to 1,400 ppb (geometric mean: 8 ppb). This average odor detection threshold range spans 5 orders of magnitude, indicating extremely wide variations from person to person. Batterman *et al.* (2023) further noted that the odor recognition threshold for hydrogen sulfide is 3 to 4 times higher than the odor detection threshold, with a range of 20-130 ppb. The nuisance threshold, which can include nausea and headaches, has been estimated to be 5 times higher than the odor detection threshold, or about 40 ppb (Batterman *et al.*, 2023).

Several inhalation regulatory exposure guidance values have been derived for hydrogen sulfide for the general population as well as for occupational settings. These values are summarized in Table 6.1. Some of these criteria were based on studies of nasal lesions (*i.e.*, physical abnormalities in the nose) in rats and mice, while others were based on the odor threshold of hydrogen sulfide. Health effects have been observed in humans only at very high, accidental exposures, or in studies with acute, controlled exposures to hydrogen sulfide in a small number of volunteers (CalOEHHA, 2008b; ACGIH, 2010; ATSDR, 2016; NIOSH, 2020). As discussed in Section 3.6, exposure guidance values are based on high-end estimates that generally over-predict health risks and are established to protect even the most sensitive individuals from potential health effects associated with exposure to the chemical for which they are derived. Often, there is no evidence for any adverse effects at exposures 10, 100, or even 1,000 times higher than these concentrations generally or for hydrogen sulfide specifically.

**Table 6.1 Inhalation Exposure Guidance Values for Hydrogen Sulfide**

Exposure Guidance Value	Exposure Period	Value	Basis	LOAEC/NOAEC	Reference
US EPA RfC <sup>a</sup>	Chronic	1.4 ppb	Nasal lesions in rats	NOAEC (10,000 ppb)	US EPA (2003a)
CalOEHHA Reference Exposure Levels	Chronic	8 ppb	Nasal changes in mice	NOAEC (30,500 ppb)	CalOEHHA (2008c)
	Acute (up to 1 hour)	30 ppb <sup>b</sup>	Controlled human exposure study	Odor threshold (12-69 ppb)	CalOEHHA (2008b)
ATSDR Minimum Risk Level	Intermediate (15-364 days)	20 ppb	Olfactory neuron loss in rats	LOAEC (30,000 ppb)	ATSDR (2016)
	Acute (1-14 days)	70 ppb	Controlled human exposure study	LOAEC (2,000 ppb)	

**Notes:**

ATSDR = Agency for Toxic Substances and Disease Registry; CalOEHHA = California Office of Environmental Health Hazard Assessment; LOAEC = Lowest Observed Adverse Effect Concentration; NOAEC = No Observed Adverse Effect Concentration; ppb = Parts Per Billion; RfC = Reference Concentration; US EPA = United States Environmental Protection Agency.

(a) For the US EPA RfC, the conversion factors and assumptions were: molecular weight (MW) = 34.08. Assuming 25°C and 760 mmHg, the NOAEL (mg/m<sup>3</sup>) = 10 ppm × 34.08 / 24.45 = 13.9 mg/m<sup>3</sup>. The NOAEL(ADJ) = 13.9 mg/m<sup>3</sup> × 6 hours / 24 hours × 7 days / 7 days = 3.48 mg/m<sup>3</sup>. The NOAEL(HEC [human equivalent concentration]) was calculated for a gas:respiratory effect in the extrathoracic region. VE(rat) = 0.19 liters/minute, VE(human) = 13.8 liters/minute, SA<sub>rat</sub> = 15 cm<sup>2</sup>, SA<sub>human</sub> = 200 cm<sup>2</sup>, RGDRET = (VE/SAET)<sub>rat</sub>/(VE/SAET)<sub>human</sub> = (0.19 / 15) / (13.8 / 200) = 0.184 = NOAEL(ADJ) × RGDR = 0.64 mg/m<sup>3</sup> (US EPA, 2003a). (b) When values were provided in units of µg/m<sup>3</sup>, I calculated the value in ppb using the following formula, using the calculation provided by NIOSH (2003) and the molecular weight provided by PubChem (NLM, 2021a): volume in liters occupied by a mole of air at 25°C and 1 atmosphere (24.45) × concentration in µg/m<sup>3</sup> / molecular weight. Thus, I calculated the CalOEHHA reference exposure level value in ppb as 24.45 × 42 µg/m<sup>3</sup> / 34.08 = 30 ppb.

Humans are exposed to hydrogen sulfide primarily by inhalation; low-level hydrogen sulfide absorption can also occur through the gastrointestinal tract. Studies in humans and experimental animals indicate that tissues with exposed mucous membranes and high oxygen demands, such as the respiratory tract, eyes, and nervous system, are the most sensitive parts of the body to hydrogen sulfide (IOMC, 2003; Guidotti, 2010; ATSDR, 2016; EC SCOEL, 2007; Batterman, *et al.*, 2023). The literature regarding the potential health effects of hydrogen sulfide is voluminous. Below, I summarize the available evidence as cited by authoritative agency assessments and published literature reviews.

## 6.1 Irritation

Irritant effects of hydrogen sulfide as a single agent at exposure levels below 20,000 ppb are not well documented in humans (EC SCOEL, 2007), although eye irritation has been reported following acute exposure to 10,000 ppb hydrogen sulfide in workers and 100,000-300,000 ppb in experimental animals (EC SCOEL, 2007). An increased prevalence of eye irritation was also reported in workers following prolonged exposure to 700-4,000 ppb hydrogen sulfide, while eye irritation was observed in experimental animals following repeated exposure to concentrations of at least 20,000 ppb hydrogen sulfide (EC SCOEL, 2007). The wide range of concentrations over which irritant effects occur are likely the result of co-exposures to other chemicals (*e.g.*, carbon disulfide) and cannot be specifically attributed to the hydrogen sulfide exposure (Batterman, *et al.*, 2023). Reported eye irritation has not resulted in permanent eye damage in humans or any microscopic effects in the eyes of experimental animals (ATSDR, 2016).

Lewis and Copley (2015) reviewed the epidemiology evidence for eye effects following lower chronic hydrogen sulfide exposures (≤10,000 ppb for over 1 year) and concluded that, although the evidence suggests irritant effects may occur in adults, the interpretation of the data is limited by a lack of exposure information, the presence of confounding co-exposures, and the potential nuisance effects attributable to

the low odor threshold of hydrogen sulfide. Furthermore, as highlighted by Batterman *et al.* (2023), studies on the ocular effects of hydrogen sulfide also provide limited spatio-temporal information, have minimal exposure contrasts, and provide limited information on biological mechanisms.

The evidence is limited regarding the potential effects of hydrogen sulfide on the skin. Peeling facial skin was reported in a single case study following acute hydrogen sulfide poisoning; however, no adverse effects on skin were noted in rodents exposed to 10,000, 30,000, or 80,000 ppb hydrogen sulfide for 6 hours/day, 5 days/week for 90 days (ATSDR, 2016).

Respiratory tract irritation was reported in rats following intermediate-duration exposures to 1,000 ppb hydrogen sulfide, while nasal lesions, which are a more severe effect than nasal irritation, were observed in rats exposed to hydrogen sulfide for acute and intermediate durations, with adverse effect levels of 80,000 ppb (3 hours/day for 5 days) and 30,000 ppb (6 hours/day, 7 days/week, for 10 weeks), respectively (ATSDR, 2016; EC SCOEL, 2007). Pulmonary edema was reported in rats following acute exposure to a very high concentration of hydrogen sulfide (400,000 ppb) for 4 hours (ATSDR, 2016).

Recent epidemiology studies have focused on communities around geothermal and volcanic sources, and around industrial sources of hydrogen sulfide. These studies are limited by a lack of reliable exposure data, a high potential for confounding co-exposures, and reporting bias (ATSDR, 2016; Lim *et al.*, 2016).

Overall, the available body of literature suggests a LOAEL of 1,000 ppb hydrogen sulfide for nose or throat irritation and a NOAEL of 10,000 ppb hydrogen sulfide for eye irritation.

## 6.2 Respiratory Effects

A review of the epidemiology evidence regarding chronic hydrogen sulfide exposure ( $\leq 10,000$  ppb for over 1 year) in occupational and community studies indicates that respiratory symptoms (*e.g.*, cough, wheezing, shortness of breath, upper respiratory infections) were the most consistently reported when compared to other types of symptoms (Lewis and Copley, 2015). These effects were temporary, however, as there was no consistent evidence of any permanent effects on the lung, such as a decrease in lung function, that would make breathing more difficult (Lewis and Copley, 2015). The reversible nature of these symptoms is also supported by case reports indicating that respiratory symptoms observed after acute, occupational exposures to hydrogen sulfide resolved within weeks to months of exposure, and by controlled human exposure studies in which there were no alterations in lung function in participants exposed to 5,000-10,000 ppb hydrogen sulfide for up to 30 minutes (ATSDR, 2016).

Respiratory distress was reported with high, acute ( $< 25$  minutes) occupational exposures to at least 40,000 ppb hydrogen sulfide, and more severe respiratory effects, such as respiratory failure and pulmonary edema, have only been reported in case studies of accidental poisonings from extremely high, acute exposures to greater than 500,000 ppb hydrogen sulfide (ATSDR, 2016).

Adverse respiratory effects have also been reported in experimental animal studies with high exposures to hydrogen sulfide. Respiratory irritation was reported in rats following intermediate-duration exposures to 1,000 ppb hydrogen sulfide, while nasal lesions, which are a more severe effect than nasal irritation, were observed in rats exposed to hydrogen sulfide for acute and intermediate durations, with adverse effect levels of 80,000 ppb (3 hours/day for 5 days) and 30,000 ppb (6 hours/day, 7 days/week for 10 weeks), respectively (ATSDR, 2016; EC SCOEL, 2007). Pulmonary edema was reported in rats following acute exposure to a very high concentration of hydrogen sulfide (400,000 ppb) for 4 hours (ATSDR, 2016).

### 6.3 Neurological Effects

Most epidemiology studies of neurological effects did not assess dose-response and/or relied on non-specific neurological measurements (Batterman *et al.*, 2023). They were also likely impacted by potential measurement bias, a wide range in odor sensitivity and psychological responses, and differing nuisance perceptions (Batterman *et al.*, 2023). Reviews of human studies that evaluated neurological effects (*e.g.*, headaches, fatigue, impaired memory, and dizziness) following chronic hydrogen sulfide exposure ( $\leq 10,000$  ppb for over 1 year) conclude that the highest quality evidence does not support a neurological-related risk in adults (Guidotti, 2010; Lewis and Copley, 2015; Lim *et al.*, 2016). Studies in communities near a source of hydrogen sulfide pollution have been conducted, but their results are not reliable because of a lack of reliable exposure data and a high potential for confounding co-exposures (ATSDR, 2016).

The only case studies that have reported neurological effects involved accidental poisonings with unknown, but likely extremely high, concentrations of hydrogen sulfide (US EPA, 2003b; IOMC, 2003; ATSDR, 2016). One controlled human exposure study reported headaches in 3 of 10 asthmatic volunteers after exposure to 2,000 ppb hydrogen sulfide for 30 minutes (ATSDR, 2016). In rat studies, decreased performance in neurological tests occurred after intermediate-duration exposure to 80,000-200,000 ppb hydrogen sulfide, and central nervous system depression occurred after acute exposure to 400,000 ppb, with unconsciousness occurring at 800,000 ppb (ATSDR, 2016).

### 6.4 Gastrointestinal Issues

Nausea and vomiting have been reported in cases of hydrogen sulfide poisoning. This has also been reported in studies of communities experiencing some episodes of higher hydrogen sulfide emissions (24-hour averages of 25-31 ppb), but these studies are not reliable because they lack reliable exposure data and have a high potential for confounding co-exposures (ATSDR, 2016).

### 6.5 Immunological Effects

There is limited evidence regarding hydrogen sulfide and immunological effects (Batterman *et al.*, 2023). Most studies are limited by a lack of hydrogen sulfide measurements, often using proximity to a particular source or odor as an exposure measure. Also, most reported immunological changes are likely the result of psychophysiological changes related to stress and sensitization, as discussed in Section 5 (Batterman *et al.*, 2023). Overall, there is no evidence for any immunological impacts of hydrogen sulfide.

### 6.6 Other Effects

The epidemiology evidence regarding cardiovascular, metabolic, and developmental effects following chronic, low exposure to hydrogen sulfide ( $\leq 10,000$  ppb for over 1 year) does not indicate a potential health hazard (Lewis and Copley, 2015; Batterman *et al.*, 2023).

Epidemiology studies of cardiovascular effects have reported inconsistent findings for multiple cardiovascular and reproductive outcomes (Batterman *et al.*, 2023). These studies have limitations that hinder their interpretation, particularly with regard to exposure estimation, co-exposures to other chemicals, and the ability to assess exposure-response relationships (Lewis and Copley, 2015). In addition, experimental animal studies indicate no effects on cardiovascular or reproductive endpoints with subchronic exposure to up to 80,000 ppb hydrogen sulfide, and no developmental effects with gestational

exposure to hydrogen sulfide, with the exception of a few studies that reported neurodevelopmental effects at exposures of 20,000 ppb and higher (ATSDR, 2016).

Metabolic studies in animals have focused on hydrogen sulfide's potential as a signaling molecule and are not generalizable to community exposures.

## 6.7 Summary

Overall, the literature regarding hydrogen sulfide toxicity indicates that it may cause effects in the respiratory tract, eyes, and nervous system, but only after inhalation exposure to very high concentrations.

Most epidemiology studies are limited by exposure measurement error and reporting or awareness bias. Regardless, these studies show that respiratory symptoms are the most common adverse effects reported at concentrations less than 10,000 ppb, but these effects are temporary, as there is no accompanying decrease in lung function. Exposure to high concentrations (>1,000 ppb) of hydrogen sulfide can also cause respiratory (including nasal) irritation in experimental animals. Eye irritation has been reported in workers, but only following prolonged exposures to hydrogen sulfide concentrations of 700 ppb and higher. Headaches have been reported in people, but only at hydrogen sulfide exposures of 2,000 ppb and higher. Severe effects have only occurred following acute exposures to extremely high concentrations of hydrogen sulfide.

## 7 Methyl Mercaptan

Methyl mercaptan is an odorous gas that is used in the manufacturing of pesticides, jet fuels, and plastics, and as an additive to natural gas. It is also a waste product in pulp and paper processing (Shertzer, 2012; ATSDR, 2014). Natural sources of methyl mercaptan include vegetation (*e.g.*, vegetables such as garlic, onions, and asparagus), animal waste, crude oils containing sulfur, and the process of microbial degradation (US EPA, 2004b; Shertzer, 2012; ATSDR, 2014). Methyl mercaptan is also produced naturally in the body by bacteria in the mouth and intestines; the generation of methyl mercaptan in the large intestine is the primary source of human exposure (US EPA, 2004b; ATSDR, 2014).

Concentrations of methyl mercaptan in outdoor air samples collected at several pulp mills were reported in the range of 0-15,000 ppb and indoor air concentrations in a construction and demolition debris recycling plant were as high as 750 ppb, with no methyl mercaptan detected in the adjacent outdoor environment (ATSDR, 2014). The odor threshold for methyl mercaptan has been reported to be in the range of 1-2 ppb (Wilby, 1969; Shertzer, 2012; Leonardos *et al.*, 1969).

There are few inhalation exposure guidance values available for methyl mercaptan for the general population, as summarized in Table 7.1. Occupational exposure guidance values are based on liver damage in rats and various acute effects at extremely high exposures in humans (accidental) and experimental animals, and the criteria for the general population are based on the occupational criteria (ACGIH, 2001; NIOSH, 1988; CARB and CalOEHHA, 2013). As discussed in Section 3.6, exposure guidance values are based on high-end estimates that generally over-predict health risks and are established to protect even the most sensitive individuals from potential health effects associated with exposure to the chemical for which they are derived. Often there is no evidence for any adverse effects at exposures 10, 100, or even 1,000 times higher than these concentrations.

**Table 7.1 Inhalation Exposure Guidance Values for Methyl Mercaptan**

Exposure Guidance Value	Exposure Period	Value	Basis	LOAEC	Reference
CalOEHHA Reference Exposure Levels	Chronic	4 ppb	Body weight decrease in rats	LOAEC (57,000 ppb)	CARB and CalOEHHA (2013); Tansy and Sherman (1981)
	Acute (up to 1 hour)	17 ppb			

**Notes:**

CalOEHHA = California Office of Environmental Health Hazard Assessment; CARB = California Air Resources Board; LOAEC = Lowest Observed Adverse Effect Concentration; ppb = Parts Per Billion.

(a) When values were provided in units of  $\mu\text{g}/\text{m}^3$ , I calculated the value in ppb using the following formula, using the calculation provided by NIOSH (2003) and the molecular weight provided by PubChem (NLM, 2021b): volume in liters occupied by a mole of air at 25°C and 1 atmosphere  $(24.25) \times$  concentration in  $\mu\text{g}/\text{m}^3$  / molecular weight. Thus, I calculated the CalOEHHA reference exposure level in ppb as  $24.45 \times 7.9 \mu\text{g}/\text{m}^3 / 48.11 = 4$  ppb.

Toxicity studies of methyl mercaptan in humans are limited to a few case reports of accidental exposures and several epidemiology studies with exposures to mixtures of chemicals including methyl mercaptan, with no information regarding estimated methyl mercaptan exposures. There have been very few studies of methyl mercaptan toxicity conducted in experimental animals.

Epidemiology studies of pulp and paper mill workers exposed to a mixture of methyl mercaptan and several other sulfur compounds, chlorine compounds, and dusts reported that the mixture of sulfur compounds was associated with headaches and respiratory effects, although lung function decrements were only reported in workers exposed to chlorine during the bleaching process of paper production (US EPA, 2004b; ATSDR, 2014). Headaches were reported when methyl mercaptan concentrations in the mixture were up to 15,000 ppb (NRC, 2013,; ATSDR, 2014). Residents of communities near pulp and paper mills reported eye and respiratory symptoms (US EPA, 2004b, 2005b). These studies do not provide evidence that methyl mercaptan exposure caused the reported effects, as exposures were to a mixture of multiple chemicals. These studies are also limited by a lack of a quantitative exposure assessment of methyl mercaptan and other sulfur compounds, the reliance on self-reported symptoms, and a lack of control for potential confounding factors.

Case reports of accidental poisonings from extremely high exposures to methyl mercaptan in occupational settings indicate that a worker who emptied tanks containing methyl mercaptan was exposed to likely an extremely high, but unknown, concentration of methyl mercaptan that resulted in coma, hemolytic anemia, methemoglobinemia, and death (NRC, 2008; Shertzer, 2012; ATSDR, 1992, 2014). Another death was reported in a worker exposed to methyl mercaptan for a few minutes at a concentration greater than 10,000,000 ppb, and a third worker at a sodium methyl sulfhydrylate factory died after an unknown level of exposure to methyl mercaptan (NRC, 2013,; ATSDR, 2014).

Studies in rats with acute exposures to methyl mercaptan indicate no adverse effects below 1,000,000 ppb. Eye and nasal irritation occurred only at exposure concentrations of at least 250,000 ppb for 4 hours; shortness of breath, ataxia, loss of righting reflex, and respiratory depression at exposure concentrations of at least 1,000,000 ppb for 1 hour; and lethargy or coma at exposure concentrations of at least 1,400,000 ppb for 15 minutes (ATSDR, 1992, 2014; NRC, 2013,). In mice, a 4-hour exposure to 114,000 ppb resulted in no clinical signs, whereas exposure to at least 258,000 ppb for the same duration resulted in shallow breathing and hypoactivity (NRC, 2013; ATSDR, 2014). In longer-term inhalation studies, rats exposed to 100,000 or 200,000 ppb methyl mercaptan for 6 hours/day for 6 or 10 days exhibited restlessness and bronchopneumonia (NRC, 2013,). Another rat study with methyl mercaptan exposures of 0, 2,000, 17,000, or 57,000 ppb for 7 hours/day, 5 days/week for 3 months reported no treatment-related effects, with the exception of reduced body weight in the rats exposed to 57,000 ppb methyl mercaptan (Tansy *et al.*, 1981).

In summary, there are no toxicity studies of humans exposed to methyl mercaptan alone, and studies in experimental animals indicate no adverse effects at repeated exposure concentrations of at least 57,000 ppb, or acute exposure concentrations of at least 250,000 ppb.

## 8 Risk Assessment

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The Consent Decree submitted in this case states that New-Indy's "average hydrogen sulfide (H<sub>2</sub>S) fenceline concentrations shall not exceed 600 ppb in any 30-minute period or 70 ppb in any seven-day period" at the fenceline (US District Court, 2022). As discussed below, based on the available data, there were a few exceedances of these values at the fenceline of the New-Indy facility (in June and September of 2021, at Station 1 only), but hydrogen sulfide levels in the surrounding community were well below these concentrations and levels that could cause health effects at all times from May 2021 to August 2023.

I evaluated the hydrogen sulfide data from each of 15 air monitoring stations located in Catawba, South Carolina (Anon, 2023a, 2023b, 2023c, 2023d). Three stations (Stations 1, 2, and 3) are located on the New-Indy plant property, and 12 stations are located outside the plant in the community (Table 8.1). No information was provided on the instrumentation used for the hydrogen sulfide analysis. One of the stations is labeled as "Collocated Accrulog<sup>2</sup> (St3)" and is co-located with one of the other stations in Table 8.1.

The hydrogen sulfide data were reported as 30-minute average concentrations in ppb and covered a monitoring period of approximately 2 years (*i.e.*, May/June 2021 to August 2023). In the dataset, non-detects were indicated with a reading of 0 or 3 ppb, with 3 ppb being the limit of detection. The majority (87-99%) of the readings at the off-site monitors were  $\leq 3$  ppb (Table 8.1).

I prepared summary statistics that include the minimum, maximum, and arithmetic mean for the 30-minute values, 1-hour average, 1-day average, 1-month average, and 1-year average for each monitor (Table 8.2). I then compared the data to the hydrogen sulfide exposure guidance values presented in Table 6.1. Specifically, I compared the 1-hour averages to the CalOEHHA REL of 30 ppb for acute exposures, the 1-day averages to the ATSDR minimum risk level (MRL) of 70 ppb for acute exposures, and the 1-month and 1-year averages to the US EPA RfC of 1.4 ppb. The results of the comparisons are presented in Table 8.3 and described below.

For the off-site monitors, the mean and maximum concentrations varied by monitoring location, for all of the averaging times. Although always well below levels that could result in health effects, these variable concentrations indicate that these low exposures varied among the proposed class members based on the geographic location of their residences, most likely due to geographic and meteorological factors.

While one cannot reliably estimate exposure for any individual without information on the location of their residence, the years they lived there, and how much time they spent at home (both indoors and outdoors), the Plaintiffs' maximum estimated exposures to the chemicals of concern were almost always below exposure guidance values and always well below exposures associated with health effects in the literature, indicating that exposures to hydrogen sulfide were not the cause of their claimed health effects.

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<sup>2</sup> I note that "Acrulog" (correct spelling) is a type of hydrogen sulfide gas monitor: <https://www.acrulog.com/products-rentals/h2s/ppm/>.

**Table 8.1 Hydrogen Sulfide Air Monitoring Stations Around the New-Indy Catawba Mill**

Area	Location	Start Date	End Date	Number of Measurements	<3 ppb H <sub>2</sub> S	
					Number	Percent
Off-site	Catawba Head Start	6/29/21	8/3/23	36,489	36,079	99%
	Liberty Hill	6/29/21	8/3/23	36,500	36,089	99%
	Millstone Creek	7/1/21	8/3/23	36,381	36,191	99%
	Riverchase Estates	6/29/21	8/3/23	36,521	36,083	99%
	Treetops	6/29/21	8/3/23	36,503	36,143	99%
	Charlotte Hwy	9/28/21	8/4/23	26,583	23,942	90%
	Indian Land	9/28/21	2/17/23	20,682	20,359	98%
	Landsford	9/27/21	8/4/23	19,099	18,865	99%
	Van Wyck	9/27/21	8/4/23	27,889	27,672	99%
	Waxhaw #1	9/27/21	12/7/21	3,427	3,421	99%
	Waxhaw #2	9/28/21	8/4/23	27,507	25,096	91%
On-site	Station 1	5/26/21	8/3/23	37,044	29,811	80%
	Station 2	5/26/21	8/3/23	37,095	34,447	93%
	Station 3	5/26/21	8/3/23	37,154	32,359	87%
	Collocated Accruiog (St3)	2/10/22	8/3/23	24,822	24,169	97%

Notes:

H<sub>2</sub>S = Hydrogen Sulfide; ppb = Parts Per Billion.

**Table 8.2 Summary of Hydrogen Sulfide Data from Air Monitoring Stations Around the New-Indy Catawba Mill (ppb)**

Location	N	30-Minute Average			1-Hour Average		
		Min	Max	Mean	Min	Max	Mean
Catawba Head Start	36,489	0	48	0.094	0	37	0.094
Liberty Hill	36,500	0	41	0.087	0	41	0.087
Millstone Creek	36,381	0	29	0.035	0	16	0.035
Riverchase Estates	36,521	0	113	0.112	0	86	0.112
Treetops	36,503	0	40	0.088	0	35	0.088
Charlotte Hwy	26,583	0	333	1.3	0	167	1.3
Indian Land	20,682	0	16	0.099	0	15	0.099
Landsford	19,099	0	167	0.084	0	83	0.084
Van Wyck	27,889	0	11	0.066	0	8.3	0.066
Waxhaw #1	3,427	0	4.7	0.019	0	4.2	0.019
Waxhaw #2	27,507	0	76	0.73	0	47	0.73
Station 1	37,044	0	1330	7.04	0	1201	7.09
Station 2	37,095	0	160	1.21	0.1	146	1.21
Station 3	37,154	0	281	1.96	0.1	251	1.97
Collocated Accruiog (St3)	24,822	0	41	0.188	0	32	0.188

Notes:

N = Sample Size; ppb = Parts Per Billion.

**Table 8.2 (Continued) Summary of Hydrogen Sulfide Data from Air Monitoring Stations Around the New-Indy Catawba Mill (ppb)**

Location	1-Day Average			1-Month Average			1-Year Average		
	Min	Max	Mean	Min	Max	Mean	Min	Max	Mean
Catawba Head Start	0	5.5	0.095	0	0.98	0.09	0.011	0.347	0.124
Liberty Hill	0	4.6	0.086	0	0.58	0.083	0.014	0.286	0.109
Millstone Creek	0	1.5	0.035	0	0.18	0.034	0.007	0.081	0.039
Riverchase Estates	0	12	0.112	0.0036	1.1	0.107	0.016	0.323	0.134
Treetops	0	4.7	0.088	0	0.98	0.08	0.015	0.28	0.11
Charlotte Hwy	0	20	1.3	0	9.3	1.5	0.13	2.9	1.2
Indian Land	0	2.6	0.10	0	0.70	0.11	0	0.14	0.047
Landsford	0	3.5	0.083	0	0.65	0.12	0.018	0.24	0.11
Van Wyck	0	1.7	0.066	0.0030	0.32	0.062	0.021	0.10	0.050
Waxhaw #1	0	0.30	0.019	0	0.040	0.012	0.019	0.019	0.019
Waxhaw #2	0	12	0.74	0.0049	2.2	0.80	0.62	0.86	0.74
Station 1	0.2	398	7.05	0.68	82	8.95	1.15	21.8	8.17
Station 2	0.2	29	1.21	0.4	5.3	1.3	0.62	2.35	1.28
Station 3	0.2	53	1.98	0.2	13	2.04	0.84	4.78	2.19
Collocated Accrulog (St3)	0	3.6	0.191	0.019	0.63	0.194	0.176	0.196	0.186

Notes:

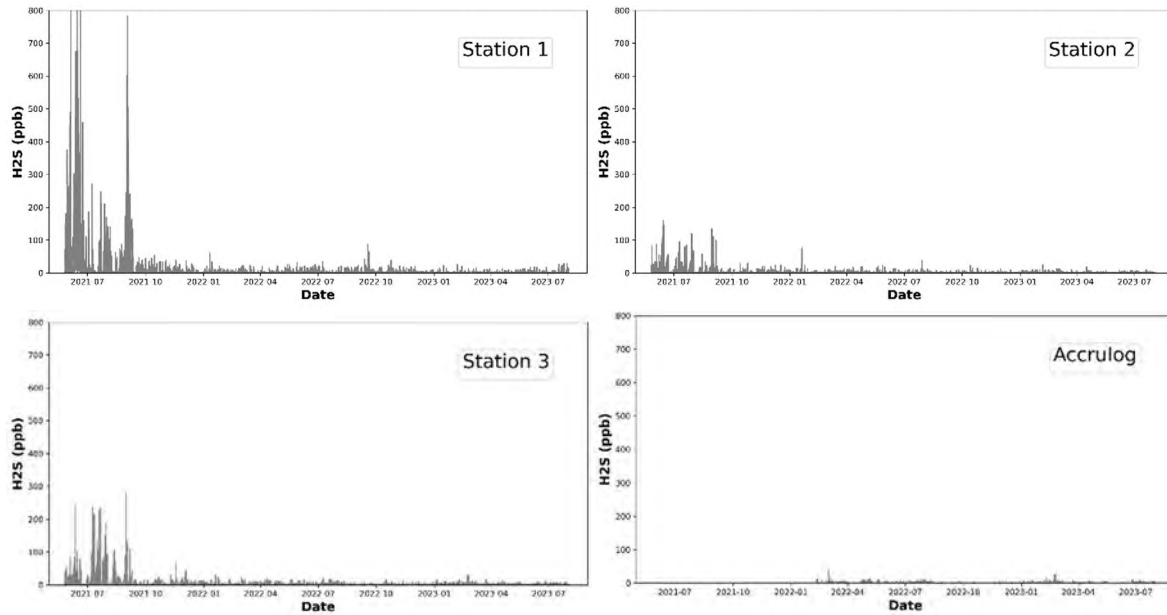
ppb = Parts Per Billion.

**Table 8.3 Comparison of Hydrogen Sulfide Concentrations Around the New-Indy Catawba Mill to Exposure Guidance Values**

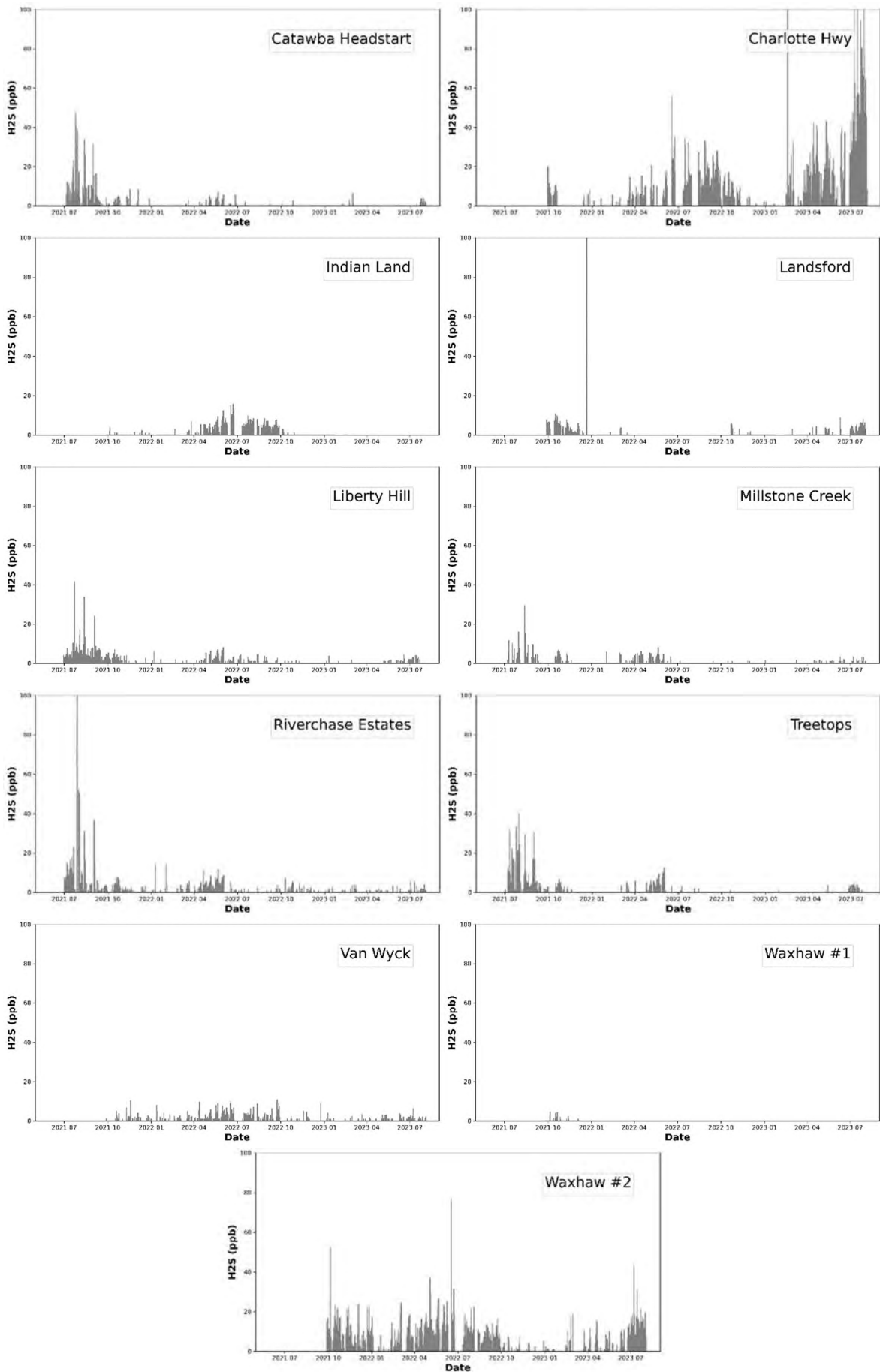
Location	N	1-Hour Avg H <sub>2</sub> S >30 ppb		1-Day Avg H <sub>2</sub> S >70 ppb		1-Month Avg H <sub>2</sub> S >1.4 ppb		Annual Avg H <sub>2</sub> S >1.4 ppb	
		#	%	#	%	#	%	#	%
Catawba Head Start	36,489	2	0.011	0	0	0	0	0	0
Liberty Hill	36,500	1	0.005	0	0	0	0	0	0
Millstone Creek	36,381	0	0	0	0	0	0	0	0
Riverchase Estates	36,521	8	0.044	0	0	0	0	0	0
Treetops	36,503	3	0.016	0	0	0	0	0	0
Charlotte Hwy	26,583	86	0.65	0	0	11	46	1	33
Indian Land	20,682	0	0	0	0	0	0	0	0
Landsford	19,099	1	0.010	0	0	0	0	0	0
Van Wyck	27,889	0	0	0	0	0	0	0	0
Waxhaw #1	3,427	0	0	0	0	0	0	0	0
Waxhaw #2	27,507	3	0.022	0	0	5	21	0	0
Station 1	37,044	691	3.64	16	2.01	18	64.3	2	67
Station 2	37,095	67	0.35	0	0	6	21.4	1	33
Station 3	37,154	186	0.98	0	0	13	46.4	1	33
Collocated Accruiog (St3)	24,822	1	0.008	0	0	0	0	0	0

Notes:

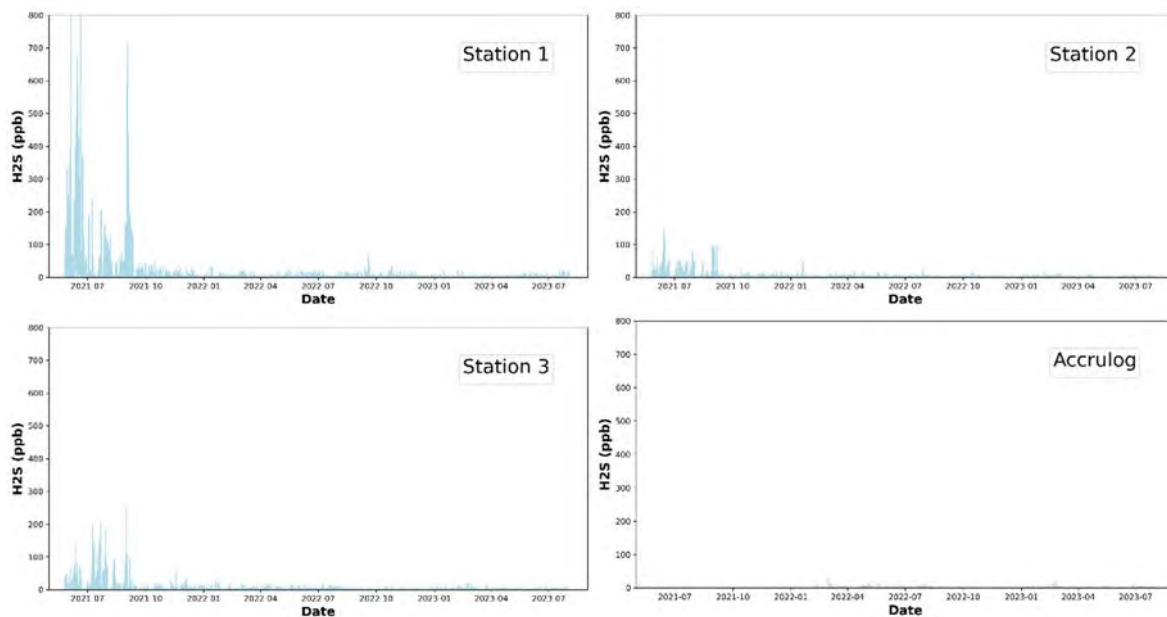
H<sub>2</sub>S = Hydrogen Sulfide; N = Sample Size; ppb = Parts Per Billion.



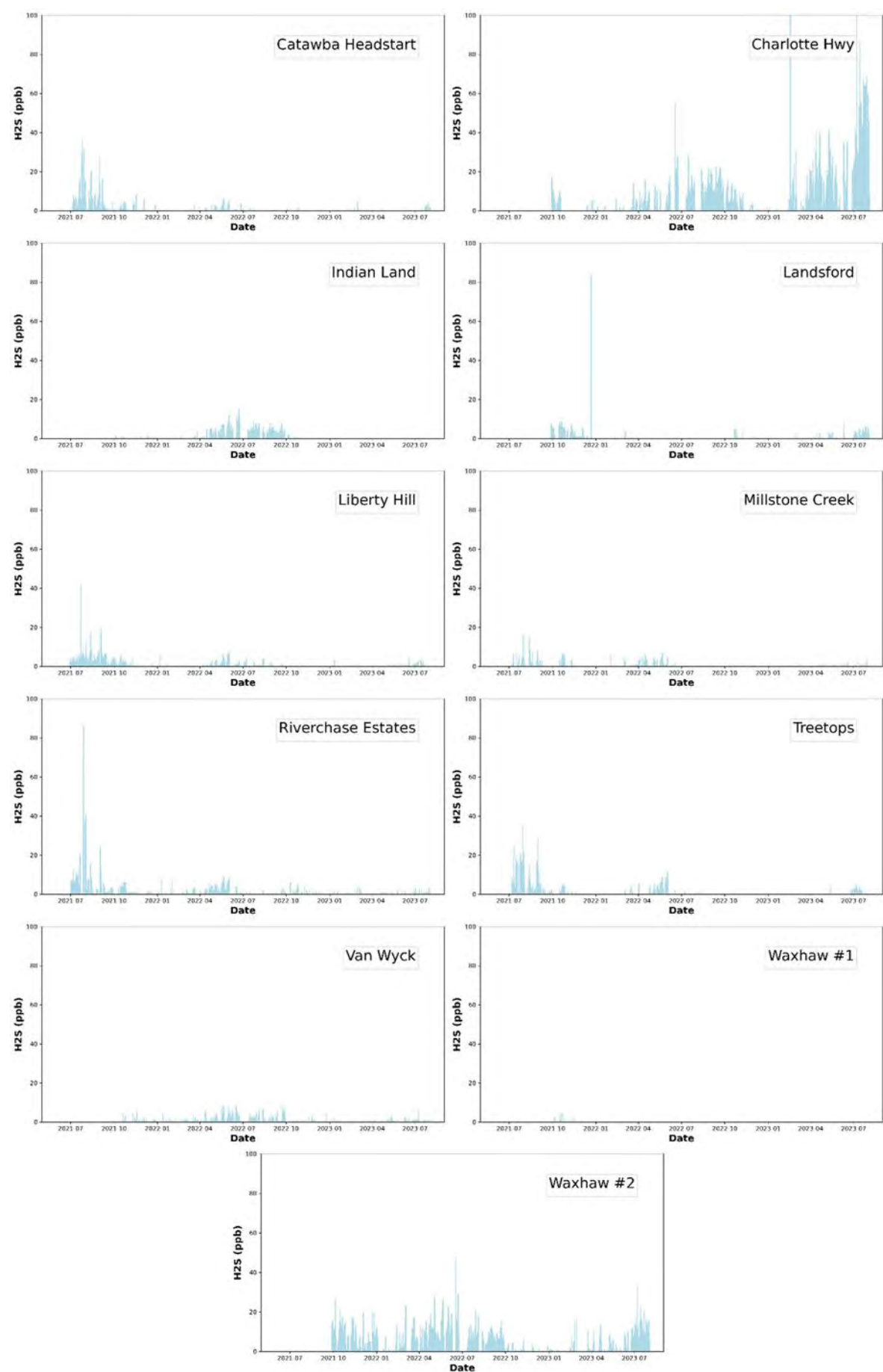
**Figure 8.1a New-Indy Catawba Mill Fenceline 30-min Average Hydrogen Sulfide Concentrations (ppb)**



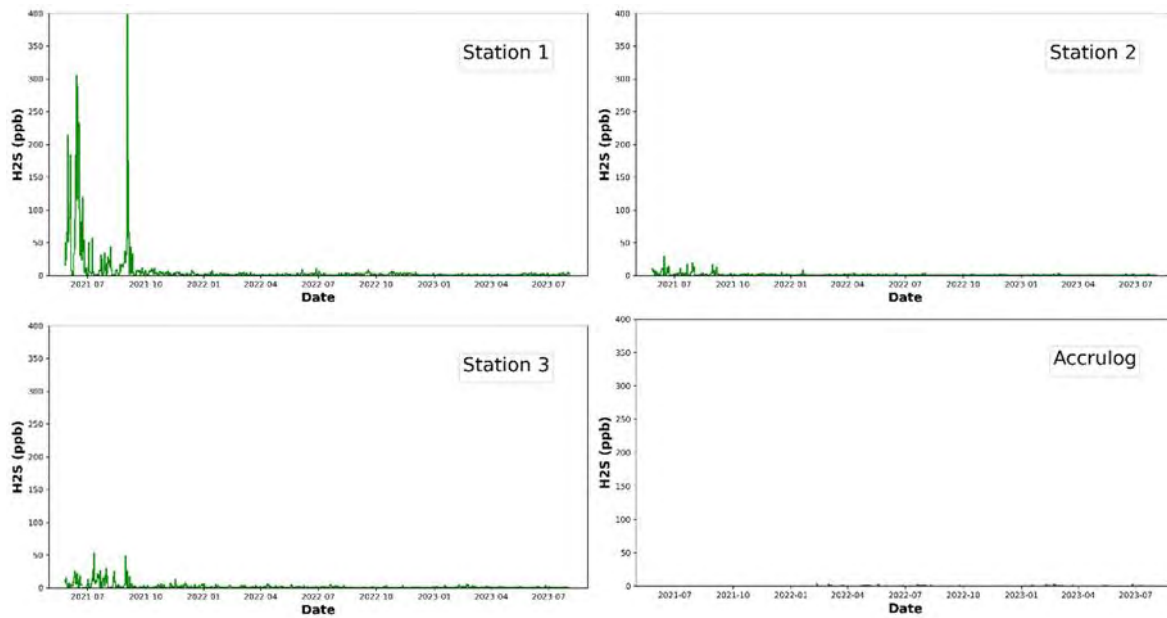
**Figure 8.1b 30-min Average Hydrogen Sulfide Concentrations (ppb) at Air Monitoring Stations Around the New-Indy Catawba Mill**



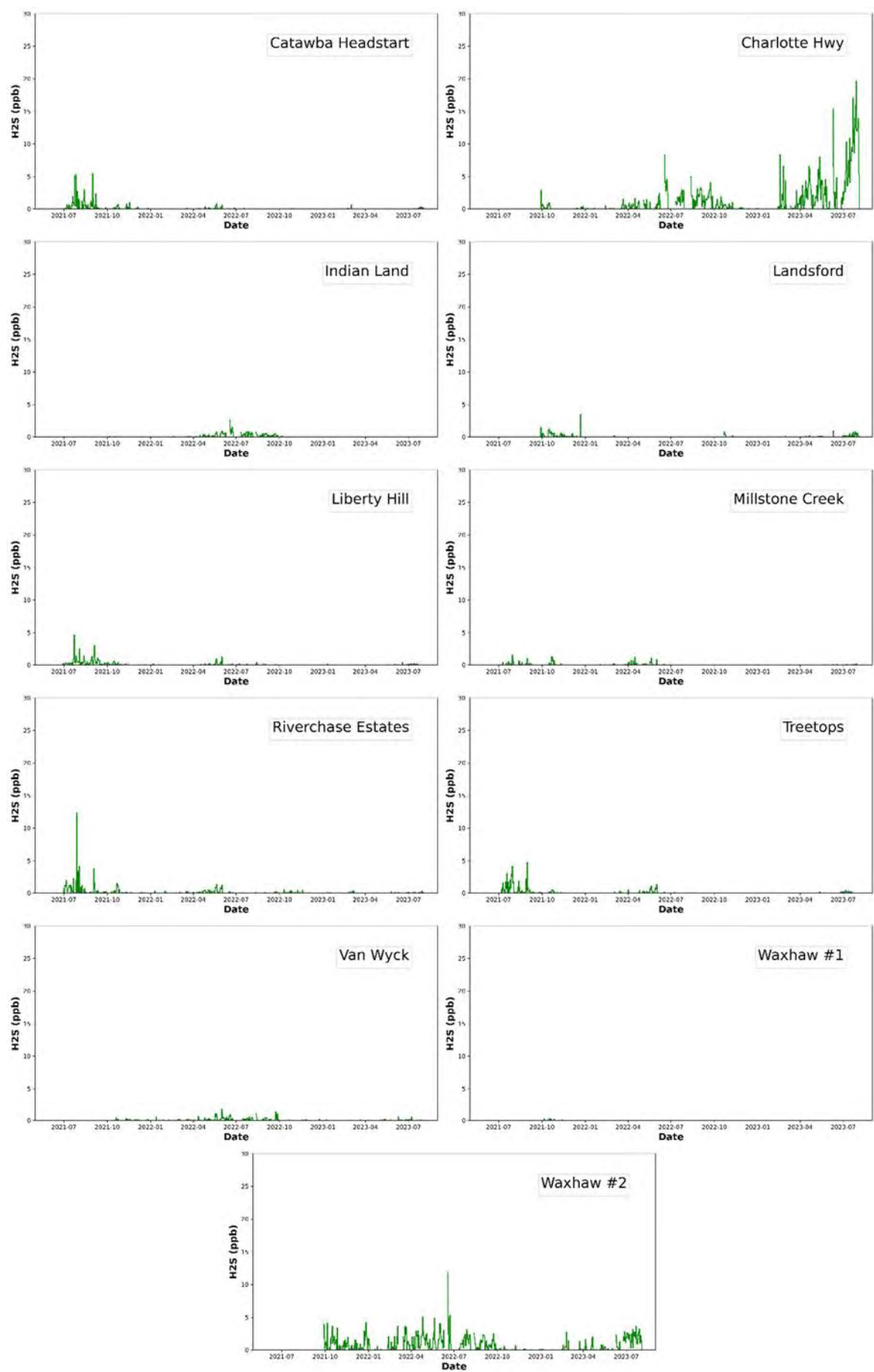
**Figure 8.2a New-Indy Catawba Mill Fenceline Hourly Average Hydrogen Sulfide Concentrations (ppb)**



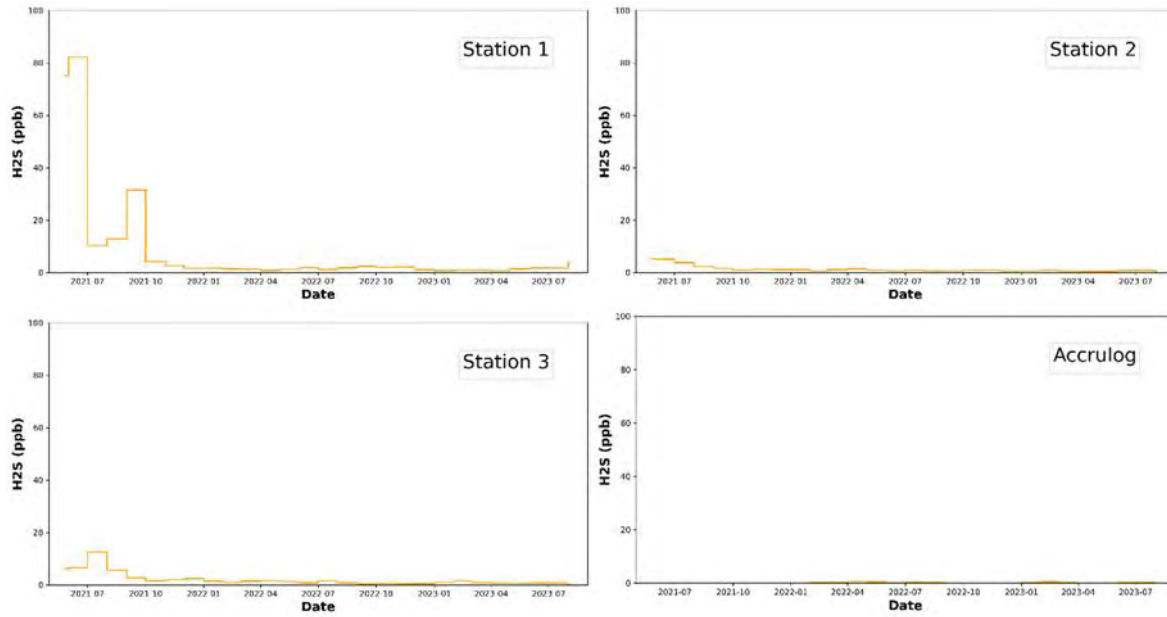
**Figure 8.2b** Hourly Average Hydrogen Sulfide Concentrations (ppb) at Air Monitoring Stations Around the New-Indy Catawba Mill



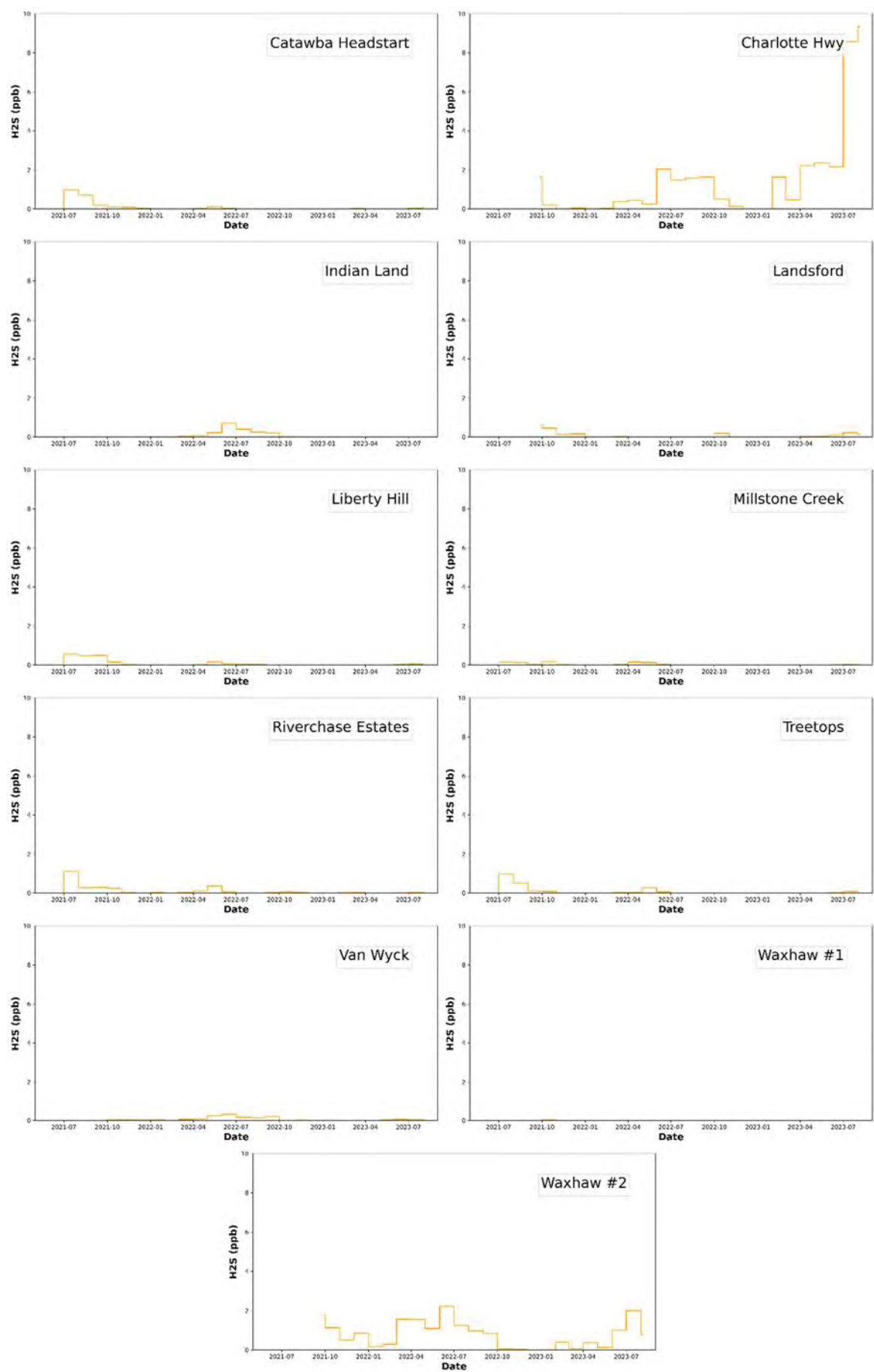
**Figure 8.3a New-Indy Catawba Mill Fenceline Daily Average Hydrogen Sulfide Concentrations (ppb)**



**Figure 8.3b Daily Average Hydrogen Sulfide Concentrations (ppb) at Air Monitoring Stations Around the New-Indy Catawba Mill**



**Figure 8.4a New-Indy Catawba Mill Fenceline Monthly Average Hydrogen Sulfide Concentrations (ppb)**



**Figure 8.4b Monthly Average Hydrogen Sulfide Concentrations (ppb) at Air Monitoring Stations Around the New-Indy Catawba Mill**

## 9 Comments on Plaintiffs' Expert Reports

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### 9.1 Dr. Allison Hecht

Dr. Hecht (2023) concluded that several of the Plaintiffs' claimed health conditions were caused by TRS, including hydrogen sulfide and other compounds, that were released from the New-Indy paper mill. As discussed in more detail below, Dr. Hecht did not consider any exposure concentrations to which the Plaintiffs may have been exposed or fully consider the exposure concentrations necessary to cause any health conditions, including those that were claimed by the Plaintiffs. She also did not consider that psychological effects are not toxic effects from odorous chemicals. She concluded that children and adolescents are more susceptible than adults to exposures, but did not provide evidence that potential exposures from the paper mill were high enough to impact children or adolescents. Finally, she concluded that a large proportion of the population was impacted by exposures based on reported health conditions in a biased sample of the population, and with no consideration of alternate causes for any condition. Dr. Hecht's conclusions are not supported by the scientific evidence, as discussed below.

#### 9.1.1 Exposure Concentrations

Dr. Hecht did not discuss any concentrations to TRS to which any Plaintiff may have been exposed. One cannot determine whether an exposure likely caused a particular health effect with no information on exposure.

#### 9.1.2 TRS and Central Nervous System Effects

Dr. Hecht (2023) stated that the impact of TRS exposure is evident based on the symptoms reported by the Plaintiffs, and opined that deficits would be indicated on neuropsychological measures. Dr. Hecht also postulated that although there would be improvements in symptoms and performance if the individuals were no longer exposed, neurological sequelae would still occur.

It is circular logic to assume that individuals were exposed based on the occurrence of symptoms, especially considering that none of these symptoms are specific to TRS and have many other known causes. Furthermore, Dr. Hecht cited several studies to support her opinion, but it is not clear how she chose these studies or whether they are representative of the scientific literature as a whole. Even setting that aside, several studies discuss exposures that are not all relevant to Plaintiffs' claimed exposures (*e.g.*, Fiedler *et al.*, 2008; Haouzi *et al.*, 2020; Hirsch, 2002; Gaitonde *et al.*, 1987; Kilburn, 1997; Kilburn *et al.*, 2010; Kilburn and Warshaw, 1995; Rumbelha *et al.*, 2016). For example, she relied on a laboratory study by Fiedler *et al.* (2008) in which participants were exposed to 0.05, 0.5, and 5 ppm hydrogen sulfide. These exposures are orders of magnitude higher than the vast majority of conc measured around site. Other studies were conducted in a community setting, and therefore individual exposures were not known (*e.g.*, Gaitonde *et al.*, 1987).

Some of the studies Dr. Hecht cited contradict her opinion. For example, Haouzi *et al.* (2020) concluded that the incidence of long-term sequelae appears to be low even in victims who require cardiopulmonary resuscitation (*i.e.*, indicating exposure to extremely high levels of hydrogen sulfide).

### 9.1.3 Emotional Symptoms

Dr. Hecht (2023) stated that "multiple studies have reported emotional concerns related to hydrogen sulfide exposure," including "continuous stress." As discussed in Section 5, there is evidence that physical symptoms can occur in certain individuals in response to an odorous chemical at exposure concentrations below its toxicity threshold as a result of stress-induced responses to perceptions of environmental risk. These symptoms are a response and not the result of a chemical-induced toxicological mechanism, so they do not indicate that the odorous chemical itself caused a toxic effect.

### 9.1.4 Children and Adolescents

Dr. Hecht (2023) stated that "[c]hildren and adolescents are also expected to have been particularly impacted by the exposures because of increased dosage of the toxins they would have received relative to adults at the same emission levels; their brains are developing; the stress associated with exposure impacts brain *[sic]*, emotional/behavioral, and academic skill development; the foul smell interferes with learning." To support this opinion, Dr. Hecht cited a case report of a 20-month-old child who was reported to have been exposed to hydrogen sulfide at levels of up to 600 ppb for up to a year (Gaitonde *et al.*, 1987). Upon admission to the hospital, the child's condition improved spontaneously and a repeat brain scan showed complete resolution of the previously seen abnormalities (Giatonde *et al.*, 1987). In addition, this exposure concentration is orders of magnitude higher than any possible exposures from the New-Indy paper mill. Importantly, even if children and adolescents are more susceptible to a similar exposure concentrations than adults, that concentration would still need to be above a certain threshold to cause any health effects in children and adolescents. Dr. Hecht has not demonstrated that any exposures were above this threshold.

### 9.1.5 Proportion of Population Impacted

Dr. Hecht concluded that "a large proportion of the population in the region of contamination" is impacted by the contamination. This conclusion is based on complaints on the South Carolina Department of Health and Environmental Control online report system and IMEs. These complaints and those who underwent IMEs do not represent an unbiased sample of the population from which they were drawn. In addition, most health conditions reported are generic and have many known causes, none of which were ruled out for any particular individual. No evidence was provided that exposure concentrations were sufficient at specific times to cause any health conditions, much less the ones reported.

## 9.2 Dr. Deborah Barsotti

Dr. Barsotti (2023) provided opinions on hydrogen sulfide, dimethyl sulfide, dimethyl disulfide, methyl mercaptan, carbon disulfide, and acetaldehyde, although her discussion of each chemical besides hydrogen sulfide was very limited. She discussed hazards and risks, but did not adequately consider these concepts in her opinions. She also discussed additive effects of exposure to multiple chemicals, but did not demonstrate that concentrations of any chemicals either alone or in combination were sufficient to cause health effects. Dr. Barsotti did not analyze data appropriately in her risk analyses. Finally, Dr. Barsotti concluded that health effects reported by Dr. Meggs were caused by emissions from the New-Indy paper mill, and these effects likely occurred in a larger portion of the surrounding population, without sufficient evidence to support this opinion.

### 9.2.1 Hazards and Risks

Dr. Barsotti included a very good description of hazard and risk, and acknowledged that risks increase with dose. However, she did not appear to fully consider dose or exposure conditions when evaluating potential health risks from pollutant exposures.

### 9.2.2 Additive Effects

Dr. Barsotti (2023) discussed additive effects when a body is exposed to two or more chemicals. As discussed below, this is not likely to be the case in the area surrounding the New-Indy paper mill.

People are typically exposed to complex mixtures of chemicals from various sources, including food, drinking water, air, and consumer products, on a daily basis (Feron and Groten, 2002). While people are not adversely affected by these near constant exposures to chemical mixtures in the environment, in some cases, simultaneous exposures to several chemicals at sufficient concentrations may result in chemical interactions that alter their toxicity. The nature of these interactions depends on the specific chemicals, their concentrations, their respective MoAs (*i.e.*, the type of mechanism that can lead to toxicity in the body), and the frequency, duration, and route of exposure to each (ATSDR, 2018b).

In general, chemical interactions may be either additive (the toxic effects produced by exposure to several chemicals at once is equal to the sum of their individual effects), synergistic (the toxic effects produced by exposure to several chemicals is equal to more than the sum of their individual effects because interactions enhance the toxicity of each chemical), or antagonistic (the toxic effects produced by exposure to several chemicals is equal to less than the sum of their individual effects because interactions diminish the toxicity of each chemical) (ATSDR, 2018b). Chemical interactions at relatively low exposure concentrations, such as those encountered in the ambient environment, usually result in effects that are additive or less than additive (Cassee *et al.*, 1998; Borgert *et al.*, 2004). For additive interactions to occur, the chemicals in a mixture must act independently and not amplify each other's toxicity.

To determine whether components of a chemical mixture are additive, one must evaluate each individual component's MoA, movement throughout the body (called pharmacokinetics), and toxicity in specific tissues of the body. In the absence of information regarding potential interactions, US EPA guidance for chemical mixture risk assessment assumes additive interactions for constituents of a mixture that have the same or a similar mode of action and/or tissue where they can exert effects (US EPA, 1989, 2000, 2007). This is a conservative approach, because assuming additivity can result in an overestimate of the risk of health effects for the chemical mixture (Cassee *et al.*, 1998).

The exposure concentrations of each component in a mixture are also the key drivers for potential synergistic effects. The available literature suggests that there is little to no likelihood of synergistic interactions between chemicals present in a mixture at low concentrations (*i.e.*, at or below their individual toxicity thresholds) typical of environmental exposures (Charles *et al.*, 2007; Crofton *et al.*, 2005; Feron *et al.*, 1995). Cedergreen (2014) reviewed the scientific literature on potential interactions among environmental chemical exposures to evaluate whether any groups of substances tend to elicit synergistic interactions and concluded that "true synergistic interactions between chemicals are rare and often occur at high concentrations." Thus, it is unlikely that exposure to mixtures of relatively low concentrations of constituents in ambient air would result in synergistic interactions.

Dr. Barsotti did not demonstrate that any concentrations of air pollutants were at sufficient concentrations to cause additive or synergistic effects. She therefore cannot predict the nature of any interactions. Given that there is little to no likelihood of synergistic interactions between chemicals present at low levels (at or

below their thresholds), which is typical of environmental exposures (*e.g.*, Charles *et al.*, 2007; Crofton *et al.*, 2005; Feron *et al.*, 1995), there is no evidence for interactions between constituents in the area.

### 9.2.3 Odors

Dr. Barsotti (2023) stated:

There are at least three mechanisms by which ambient odors may produce health symptoms. First, symptoms can be induced by exposure to chemicals with odor properties at levels that also cause irritation or other toxicological effects. That is, irritation rather than the odor is the cause of the health symptoms. Second, health symptoms from odorants at non-irritant concentrations can be due to innate (genetically coded) or learned aversions. Third, symptoms may be due to a co-pollutant.

It is not entirely clear what she meant by the first mechanism. I assume she meant that the chemical can be present at a concentration that is toxic, rather than the odor itself causing effects. Every chemical is toxic at some minimum exposure concentration (see Section 3.2). It appears that the second mechanism refers to psychological responses, rather than toxicological responses (see Section 5). The third refers to another chemical altogether. Dr. Barsotti did not show that any potential exposures associated with the New-Indy paper mill were sufficient to cause any toxic effects, or were correlated with other chemicals at sufficient doses to cause toxic effects.

### 9.2.4 Hazard Profiles

Dr. Barsotti (2023) provided a table with health-based air benchmarks for hydrogen sulfide, dimethyl sulfide, dimethyl disulfide, methyl mercaptan, carbon disulfide, and acetaldehyde. She did not describe the studies on which these values were based or, importantly, indicate the averaging time for any of the standards. She also briefly described hazards associated with these chemicals, but did not indicate the exposure conditions under which any health conditions may occur or whether concentrations in the area near the New-Indy paper mill exceeded those concentrations.

### 9.2.5 Risk Analysis

Dr. Barsotti (2023) conducted a risk assessment for hydrogen sulfide. She first compared 30-minute concentrations to thresholds of 5 and 600 ppb with no scientific rationale for doing so. She then indicated that residents in the vicinity of the New-Indy paper mill would be able to smell hydrogen sulfide/TRS, but as discussed above, smelling an odor does not mean that the chemical causing that odor is at a sufficient concentrations to cause any toxicological effects. She also compared air dispersion modeling and indicated the overall maximum 30-minute average concentration was  $>900 \mu\text{g}/\text{m}^3$  (*i.e.*, 600 ppb). The ATSDR acute MRL is specifically for 1- to 14-day exposures. Therefore, it is not appropriate to compare 30-minute exposures to the MRL. As noted in Section 8, there were no 1-day average hydrogen sulfide concentrations  $>70$  ppb at monitors in the area near the New-Indy paper mill, and only 16 exceedances (out of a total of 37,044 samples) in one of the three fenceline monitors.

Dr. Barsotti also mentioned effects occurring at less than 30 ppb based on a study by Campanga *et al.* (2004). In this ecological study, the authors observed that for children, but not adults, previous-day hydrogen sulfide concentrations were positively associated with hospital visits for respiratory diseases. Fixed community monitors were used to measure ambient hydrogen sulfide concentrations, which likely resulted in exposure measurement error. In addition, co-exposures were not fully accounted for and

outcome misclassifications in hospitals were possible, and some people might not have sought treatment at the target hospitals in the study.

Dr. Barsotti also discussed effects from hydrogen sulfide odors, but the effects she discussed are psychological effects, not toxic effects from the chemicals causing the odors.

### 9.2.6 Conclusions

In her conclusions, Dr. Barsotti (2023) concluded that health effects reported by Dr. Meggs were caused by emissions from the New-Indy paper mill, and these effects likely occurred in a larger portion of the surrounding population. Dr. Barsotti did not demonstrate that concentrations of hydrogen sulfide were sufficient to cause any health conditions, much less those listed by Dr. Meggs. Dr. Barsotti's conclusions are not supported by the scientific evidence.

## 9.3 Dr. Harold Palevsky

Dr. Palevsky's discussed hydrogen sulfide "and its potential for upper and lower respiratory tract (pulmonary) toxicity" (Palevsky, 2023). He concluded that questionnaires and IMEs showed symptoms "temporally related to and provoked by the noxious odors coming from the New Indy plant" and that a large segment of the population exposed experienced certain pulmonary issues as a result of emitted hydrogen sulfide (Palevsky, 2023).

Dr. Palevsky did not discuss the hydrogen sulfide exposure dose, duration, or frequency necessary to cause any health conditions. He also did not make any comparisons of measured concentrations in the area around the New-Indy paper mill to those that may result in health conditions. He noted some conditions were likely a result of odors, but did not indicate that those conditions were from toxic effects of the odorous chemicals.

Dr. Palevsky provided no evidence that all residents in the area have likely experienced either exposures sufficient to cause health conditions or any symptoms associated with exposures. He also did not consider that the ability to detect and recognize odors varies considerably, as discussed in Section 5, as do any symptoms that an individual may experience in response to odors.

## 9.4 Dr. William Fee

Dr. Fee, an otolaryngologist, noted that "low concentrations" of the chemicals can "cause mucous membrane irritations, headaches, sleep disturbances, fatigue, and or quality of life [*sic*]" (Fee, 2023). He concluded that, based on his evaluation of six Plaintiffs, "a larger segment of the population is likely to have similar problems" (Fee, 2023). There is no evidence that these six people are similar to or representative of population as a whole with respect to exposures or symptoms.

Similar to Dr. Meggs, as discussed below, Dr. Fee did not evaluate possible alternative causes for the Plaintiffs' claimed health effects.

To support his opinions, Dr. Fee relied on only one study, by Bates *et al.* (1998), who evaluated geothermal emissions in a community in Rotorua, New Zealand. Dr. Fee stated that Bates *et al.* (1998) "found a higher incidence ratio of cancers of the trachea, bronchus and lung in Maori women." To the contrary, Bates *et al.* (1998) concluded, "There are inadequate exposure data for Rotorua to permit conclusions on likely causal associations."

## 9.5 Dr. William Meggs

In addition to IMEs, Dr. Meggs discussed health effects associated with hydrogen sulfide and other chemicals. He also discussed US EPA's hydrogen sulfide standard. He stated that hydrogen sulfide can cause "horrific danger to humans exposed to even low levels," but provided no evidence to support this. He indicated that this is supported by the US EPA RfC of 1 ppb, and said that standards do not apply to sensitive populations. In fact, US EPA's definition of an RfC is:

An estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (**including sensitive subgroups**) that is likely to be without an appreciable risk of deleterious effects during a lifetime. It can be derived from a NOAEL, LOAEL, or benchmark concentration, with uncertainty factors generally applied to reflect limitations of the data used. Generally used in EPA's noncancer health assessments. [emphasis added] (US EPA, 2002a)

That is, by definition, the RfC protects sensitive populations. Moreover, the hydrogen sulfide RfC is based on the highest dose at which no nasal lesions were reported in rats, and includes a composite UF of 300. This indicates that exposures even orders of magnitude above the RfC are not likely associated with health effects.

### 9.5.1 Epidemiology Studies

Dr. Meggs (2023) discussed two community studies (Campagna *et al.* [2004] and Jaakkola *et al.* [1990]), which he suggested provide evidence for hydrogen sulfide causing effects above the ATSDR MRL. Because they are community studies, they do not have information on individual exposures and therefore are not informative with respect to causation. The ecological study by Campagna *et al.* (2004) is discussed above in Section 9.2.5.

In the study by Jaakkola *et al.* (1990), participants from a "severely polluted" community had statistically significant elevated odds of reporting nasal and eye symptoms and cough compared to participants from "non-polluted" community. Issues with study quality raise questions regarding study validity. Because of the mixed exposures, the role of hydrogen sulfide (if any) is unclear. The authors explained the study aim to participants, which may have caused over-reporting of symptoms. A cross-sectional, self-administered questionnaire was used to gather data on the occurrence (*i.e.*, often or constantly) of a variety of respiratory symptoms and effects during two time periods (the past 4 weeks and the previous 12 months). No objective measures of health status were taken. Because all symptoms were self-reported, there is potential for reporting bias due to concerns about pollution.

He also cited a study by Legator *et al.* (2001) as evidence of effects from chronic low-level exposures to hydrogen sulfide. In this study, participants from two exposed communities self-reported increased occurrences of health symptoms compared to participants from three reference (non-exposed) communities. Specifically, there were increased odds of symptoms in the exposed groups compared to the unexposed group. The highest odds ratios were for central nervous system, respiratory, and blood symptoms. However, there were inconsistent and unreliable hydrogen sulfide exposure data for Puna (exposed city). In addition, potential issues with participant selection also create uncertainties in the study results. Participants from Odessa (exposed city) were originally plaintiffs of a lawsuit ongoing in the community. If the lawsuit was related to environmental pollution, then it could be a potential source of bias (*i.e.*, the exposed group likely over-reported symptoms). Recall bias and co-exposures may also have been issues.

### 9.5.2 Mixtures

To address the Plaintiffs' possible exposures to methyl mercaptan, dimethyl sulfide, and carbon disulfide, Dr. Meggs provided only a sentence or two for each chemical. The scientific literature on these substances is vast and he did not describe the pertinent exposure conditions for potential health effects. Dr. Meggs also stated that the Plaintiffs were exposed to TRS as "complex mixtures with synergistic toxicity." Chemical mixtures are discussed in Section 9.2.2.

### 9.5.3 Air Monitoring

As discussed in Section 8, the air monitoring data do not indicate that health effects would be expected to occur.

### 9.5.4 Independent Medical Examinations

Dr. Meggs (2023) conducted IMEs of the Plaintiffs virtually (*i.e.*, did not examine them in person). He concluded that their claimed exposures to hydrogen sulfide and TRS emissions "caused increased illnesses in the exposed population, which includes, but is not limited to, the class representatives" (Meggs, 2023). He did not discuss the exposure dose, frequency, and duration of exposure for any Plaintiff. He also did not review the Plaintiffs' medical records or consider possible alternative causes for their claimed health effects. Thus, his conclusion is not supported by the scientific evidence.

## 10 Conclusions

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I conclude to a reasonable degree of scientific certainty:

- The vast majority of the Plaintiffs' alleged physical effects are common conditions in the general population with many known risk factors.
- Odor itself is not an indicator of toxicity or adverse health effects. Some of the alleged conditions have been reported as responses to the detection of an unpleasant odor, but these are not the result of a toxic effect of the odorant.
- Concentrations of hydrogen sulfide in the area around the New-Indy paper mill are below exposures associated with health effects in the literature.
- There are no toxicity studies of humans exposed to methyl mercaptan alone. Studies in experimental animals indicate no adverse effects of methyl mercaptan at repeated exposure concentrations of at least 57,000 ppb or acute exposure concentrations of at least 250,000 ppb.
- The Plaintiffs' maximum estimated exposures to hydrogen sulfide were almost always below exposure guidance values and always well below exposures associated with health effects in the literature, indicating that any exposures to hydrogen sulfide were not the cause of any of the Plaintiffs' claimed health effects.
- Dr. Hecht, Dr. Barsotti, Dr. Palevsky, Dr. Fee, and Dr. Meggs did not adequately evaluate the state of the science regarding potential health effects from exposure to hydrogen sulfide and other chemicals, or whether the science supports general causation. They did not demonstrate that hydrogen sulfide concentrations near the New-Indy paper mill were sufficient to cause any health conditions at all, much less in Plaintiffs.

In conclusion, based on my review of case-specific documents and publicly available scientific literature, I conclude to a reasonable degree of scientific certainty that Plaintiffs' alleged health conditions were not attributable to exposures to TRS, including hydrogen sulfide or methyl mercaptan, near the New-Indy paper mill.

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# **Appendix A**

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## **Curriculum Vitae of Julie E. Goodman, Ph.D., DABT, FACE, ATS**



**Julie E. Goodman, Ph.D., DABT, FACE, ATS**  
**Principal**

jgoodman@gradientcorp.com

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**Areas of Expertise**

Epidemiology, toxicology, systematic review, evidence integration, meta-analysis, carcinogenesis, dose-response analysis, product safety, risk assessment, risk communication.

**Education & Certifications**

Ph.D., Environmental Health Sciences/Toxicology, Johns Hopkins Bloomberg School of Public Health, 2002

Sc.M., Epidemiology, Johns Hopkins Bloomberg School of Public Health, 2000

S.B., Environmental Engineering, Massachusetts Institute of Technology, 1996

Diplomate, American Board of Toxicology (DABT), 2005; recertified 2010, 2015, 2020

Fellow, American College of Epidemiology (FACE), 2014

Fellow, Academy of Toxicological Sciences (ATS), 2014; recertified 2019

**Professional Experience**

2004 – Present GRADIENT, Boston, MA

Principal. Evaluate toxicity and epidemiology data in the context of causation analysis and human health risk assessments. Focus on substances in consumer products, pharmaceuticals, and medical devices, and chemicals in the workplace and the environment.

2009 – 2017 HARVARD T. H. CHAN SCHOOL OF PUBLIC HEALTH, Boston, MA

Adjunct Faculty Member. Department of Epidemiology. Co-instructor of course entitled, "Research Synthesis & Meta-analysis."

2002 – 2004 NATIONAL CANCER INSTITUTE, Bethesda, MD

Cancer Prevention Fellow. Conducted a number of molecular epidemiology studies analyzing the relationships between inflammatory gene polymorphisms and colon cancer risk. Instrumental in the development of a powerful statistical tool for cancer risk assessment.

**Continuing Education Courses and Other Training**

- Introduction to Open-Access Computational Toxicology Tools (web course), Society of Toxicology 2020 Annual Meeting, April 2020
- Protecting Human Research Participants Online Course, National Institutes of Health (NIH) Office of Extramural Research, 2015
- Tools and Technologies in Translational Toxicology, Society of Toxicology 2013 Annual Meeting, San Antonio, TX, March 2013

9/14/2023

**Julie E. Goodman, Ph.D., DABT, FACE, ATS**

- Use of Expert Elicitation to Inform Decisionmaking, Society for Risk Analysis 2012 Annual Meeting, San Francisco, CA, December 2012
- Novel Statistical Challenges in Environmental Epidemiology Workshop, 3<sup>rd</sup> North American Congress of Epidemiology, Montreal, Canada, June 2011
- Comparative Biology of the Lung, Society of Toxicology 2010 Annual Meeting, Salt Lake City, UT, March 2010
- Introduction to the Benchmark Dose Methodology and Interactive Application of United States Environmental Protection Agency (US EPA) Benchmark Dose Software (BMDS), Version 2.1, Society for Risk Analysis 2010 Annual Meeting, Salt Lake City, UT, December 2010
- Green Innovation for Business Conference (Moderator, Green Chemistry and Greenwashing Workshops), Boston, MA, June 2009
- Decision-making for Recommendations and Communication Based on the Totality of Food-related Research, International Life Sciences Institute Workshop, Washington, DC, December 2008
- 2008 Board of Health Certification Program, Massachusetts Association of Health Boards, Marlborough, MA, November 2008
- What is Evolutionary Epidemiology? American College of Epidemiology Annual Meeting, Tucson, AZ, September 2008
- Research Ethics in Studying Genes and the Environment in Diabetes Among Ethnic Minorities, American College of Epidemiology Annual Meeting, Tucson, AZ, September 2008
- Use of Data for Development of Uncertainty Factors in Non-Cancer Risk Assessment, Society of Toxicology 47<sup>th</sup> Annual Meeting, Seattle, WA, March 2008
- International Society of Regulatory Toxicology and Pharmacology Workshop: Conducting and Assessing the Results of Endocrine Screening, Bethesda, MD, February 2008
- Assessment of Abuse Liability and Physical Dependence, Northeast Chapter Society of Toxicology Annual Meeting, Groton, CT, October 2007
- Practical Issues and Procedures for Preclinical Safety Testing, 2007 BioReliance Toxicology Technical Seminars, Boston, MA, October 2007
- Introduction to Pharmacoepidemiology: Practical Applications and Analytic Methods, American College of Epidemiology 25<sup>th</sup> Annual Meeting, Ft. Lauderdale, FL, September 2007
- SAS Programming I: Essentials, SAS Institute, Boston, MA, July 2007
- Introduction to Bayesian Modeling of Epidemiologic Data, Society for Epidemiologic Research 40<sup>th</sup> Annual Meeting, Boston, MA, June 2007
- Systematic Review and Meta-analysis, Society for Epidemiologic Research 40<sup>th</sup> Annual Meeting, Boston, MA, June 2007
- The Biology and Toxicology of the Peri- and Post-natal Development, Society of Toxicology 46<sup>th</sup> Annual Meeting, Charlotte, NC, March 2007
- Reproductive Toxicity Testing: Study Designs, Evaluation, Interpretation, and Risk Assessment, Society of Toxicology 45<sup>th</sup> Annual Meeting, San Diego, CA, March 2006
- Project Managers Bootcamp I, PSMJ Resources, Inc., Cambridge, MA, April 2005
- Development and Interpretation of Toxicokinetic Data for Risk and Safety Assessment, Society of Toxicology 44<sup>th</sup> Annual Meeting, New Orleans, LA, March 2005
- Survival Analysis, Graduate Summer Institute of Epidemiology and Biostatistics, Johns Hopkins Bloomberg School of Public Health, Baltimore, MD, June 2003
- Speaking on the Job, Cancer Prevention Fellowship Program, National Cancer Institute (NCI), Rockville, MD, February 2003
- Grants and Grantsmanship Workshop, Cancer Prevention Fellowship Program, NCI, Rockville, MD, January 2003
- Spotted Gene Expression Microarray Workshop, Advanced Technology Center, NCI, Gaithersburg, MD, October 2002
- Laboratory of Cellular Carcinogenesis and Tumor Promotion/Laboratory of Human Carcinogenesis/Laboratory of Experimental Carcinogenesis Interlaboratory Seminar, Monthly, 2002-2004
- Division of Cancer Prevention, Office of Preventive Oncology Colloquia Series on Cancer Prevention Topics, Weekly, 2002-2004

**Julie E. Goodman, Ph.D., DABT, FACE, ATS**

- Radiation Safety for Authorized Users, NIH Radiation Safety Branch, Bethesda, MD, Sept. 2002
- Molecular Prevention Course, NCI Summer Curriculum in Cancer Prevention, Rockville, MD, August 2002
- Principles & Practice of Cancer Prevention and Control Course, NCI Summer Curriculum in Cancer Prevention, Rockville, MD, July-August 2002

**Professional Activities**

- Philanthropy Liason, National Charity League, 2023-Present
- Member, Evidence-based Toxicology Collaboration (EBTC) Interim Scientific Advisory Council (iSAC), November 2021-Present
- Chair, Communications Committee, Academy of Toxicological Sciences, 2021-2022
- Member, Board of Directors, Academy of Toxicological Sciences, 2020-2023
- Member, Communications Committee, Academy of Toxicological Sciences, 2020-2021
- Canton, Massachusetts, COVID Task Force, 2020-Present
- Scientific Advisory Board Member, National Stone, Sand, and Gravel Association, 2019-Present
- Invited Lecturer, "Introduction to Meta-analysis," Northeastern College of Professional Studies, May 8, 2019
- Invited Participant, "Excellence in Risk Analysis," Society for Risk Analysis (SRA) Workshop, June 2018
- Reviewer, R21 Hurricane Applications, National Institutes of Health (NIH), January 2018
- Member, Regis College Doctoral Thesis Committee, 2015-2017
- Reviewer, R21 NIH Exploratory/Developmental Research Grant Proposal, NIH, September 2017
- Invited Panelist, Cancer Prevention Fellowship Program (CPFP) Alumni Career Panel, September 2016
- Reviewer, K99 Career Research Grant Proposal, NIH, July 2016
- Invited Lecturer, "An Introduction to Meta-analysis," Johns Hopkins Bloomberg School of Public Health, April 5, 2016
- Member, Scientific Advisory Council, Evidence-based Toxicology Collaboration (EBTC) at Johns Hopkins Bloomberg School of Public Health, December 2015-November 2021
- Invited Observer, *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: Volume 113: Some Organochlorine Insecticides and Some Chlorophenoxy Herbicides*, Lyon, France, June 2015
- Mentor, Society of Toxicology Mentor Match Program, January 2015-Present
- Member, Speaker Bureau, Society of Toxicology, July 2014-2016
- Chair, "Implementing NRC Recommendations: IRIS," SRA Annual Meeting, 2014
- Member, California Breast Cancer Research Program (CBCRP) Chemicals Testing and Occupational Exposures Review Panel, November 2014
- Proposal Reviewer, Scientific Panel, California Breast Cancer Research Program (CBCRP), June 2014
- Invited Epidemiology Panel Member, The International Council of Chemical Associations Long-Range Research Initiative and Joint Research Centre Workshop, "What is Safe? Integrating Multi-Disciplinary Approaches for Decision Making about the Human Health and Environmental Impacts of Chemicals." Lugano, Switzerland, June 2014
- Proposal Reviewer, John Templeton Foundation, 2014
- Co-Chair, "Understanding Weight of Evidence: Exploring Different Approaches to Integrating Evidence from Diverse Data Streams," Society of Toxicology, 2014
- Co-Chair, "Epidemiology for Toxicologists: What the Numbers Really Mean," Society of Toxicology, 2014
- Best Paper Awards Selection Committee, Risk Assessment Section, Society of Toxicology, 2014
- Peer Reviewer, Provisional Peer-Reviewed Toxicity Values for Styrene-Acrylonitrile (SAN Trimer), US EPA Draft Document, December 2013
- Keynote Speaker and Scientific Committee Member, Isocyanates & Health Conference, April 2013
- Presidential Task Force Member, Strategic Plan, American College of Epidemiology, 2013
- Proposal Reviewer, National Science Foundation, 2013

**Julie E. Goodman, Ph.D., DABT, FACE, ATS**

- Invited Participant, ILSI Health and Environmental Sciences Institute Emerging Issue Workshop: Evaluating Causality in Epidemiology, October 2012
- Invited Panel Member, "Using Mode of Action to Support the Development of a Multipollutant Science Assessment," US EPA Workshop, May 2012
- Editorial Board Member, *Carcinogenesis*, 2012-2014
- Invited Participant on "Improving Science-Based Regulation," The George Washington University Regulatory Studies Center and the Center for Risk Science and Public Health, January 2012
- Member, Massachusetts Environmental Justice Assistance Network, 2010-Present
- Board Member, American College of Epidemiology, 2011-2013
- Nominating Committee, Society of Toxicology, 2009-2011
- Elected Member, Canton, Massachusetts Board of Health, 2008-Present
- Editorial Board Member, *The Open Biomarkers Journal*, 2008-Present
- Managing Editor, *Journal of Environmental Protection Science*, 2008-2010
- Member, Canton, Massachusetts Medical Reserve Corps, 2007-Present
- Peer Reviewer, *Texas Commission on Environmental Quality, Development Support Document for Nickel and Inorganic Nickel Compounds, Preliminary Draft*, May 2009
- Invited Observer, *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: Volume 100, Meeting C: Metals, Particles and Fibres*, Lyon, France, March 2009
- Abstract Awards Selection Committee, Risk Assessment Section, Society of Toxicology, 2008
- Secretary/Treasurer, Risk Assessment Specialty Section, Society of Toxicology, 2007-2009
- Editorial Board Member, *Journal of Environmental Protection Science*, 2007-2008
- Guest Lecturer, Cancer Epidemiology, University of Maryland, 2004
- Member, Cancer Prevention Fellowship Scientific Education Committee, NCI, 2003
- Guest Lecturer, Xenobiotic Metabolism, Johns Hopkins Bloomberg School of Public Health, 2001-2002
- Reviewer: *African Journal of Biotechnology; American Journal of Ophthalmology; American Journal of Pathology; Annals of Epidemiology; Applied Economics Letters; Biomarkers & Prevention; Cancer Epidemiology; Cancer Genetics and Cytogenetics; Cancer Research; Carcinogenesis; Chemical Research in Toxicology; Chemico-Biological Interactions; CHEST; Clinical Cancer Research; Critical Reviews in Toxicology; Environmental Health Perspectives; Environment International; Environmental Science & Technology; Epidemiology; Food Science & Nutrition; Global Epidemiology; Human and Experimental Toxicology; Inhalation Toxicology; International Journal Of Environmental Health Research; International Journal of Environmental Research and Public Health; Journal of Cellular Biochemistry; Journal of Clinical Epidemiology; Journal de Pediatria; Journal of Exposure Science and Environmental Epidemiology; Journal of Human and Ecological Risk Assessment; Journal of Occupational and Environmental Medicine; Journal of Toxicology and Environmental Health, Part A: Current Issues; Medical Journal of Australia; NeuroToxicology; PeerJ; Pharmacogenetics; Preventive Medicine Reports; Regulatory Toxicology and Pharmacology; Risk Analysis; Safety and Health at Work; Scientific Reports (Nature Publishing Group); Toxicology; Toxicology and Applied Pharmacology; Toxicological Sciences*

**Honors and Awards**

- Best Poster Award, Environment, Health & Safety Poster Session, Polyurethanes Technical Conference, October 2018
- Distinguished Alumna Award, Johns Hopkins University, April 2015
- Chauncey Starr Distinguished Young Risk Analyst Award, Society for Risk Analysis, 2014
- Best Overall Abstract, Risk Assessment Specialty Session, Society of Toxicology, San Antonio, TX, 2013
- International Dose-Response Society Outstanding New Investigator Award, 2012
- Top 10% Best Overall Abstracts in Risk Assessment, Risk Assessment Specialty Section, Society of Toxicology, Seattle, WA, 2008
- Fellows Award for Research Excellence: \$1,000 Travel Award, National Institutes of Health, Bethesda, MD, 2004

**Julie E. Goodman, Ph.D., DABT, FACE, ATS**

- Honorable Mention Poster Presentation, Center for Cancer Research 4<sup>th</sup> Annual Fellows and Young Investigators Retreat, Williamsburg, VA, March 2004
- Graduate Student Travel Award, Gordon Research Conference on Hormonal Carcinogenesis, 1999, 2001
- Travel Award, Third World Congress on Alternatives and Animal Use in the Life Sciences, Bologna, Italy, 1999
- Howard Hughes Predoctoral Fellowship Award, 1997-2002
- NIEHS Training Grant Graduate Fellowship Award, Johns Hopkins Bloomberg School of Public Health, Baltimore, MD, 1996-1997
- Tau Beta Pi, National Engineering Honor Society, 1995-1996
- Chi Epsilon, National Civil Engineering Honor Society, 1994-1996

**Professional Affiliations**

American College of Epidemiology; Society for Risk Analysis; Society for Risk Analysis New England Chapter; Society of Toxicology; American Board of Toxicology; Academy of Toxicological Sciences; International Dose-Response Society

**Selected Projects**

Trade Association: Conducted a systematic review of gas cooking or indoor nitrogen dioxide (NO<sub>2</sub>) and asthma or wheeze in children.

Food company: Reviewed applicable US FDA requirements for new dietary ingredients and proposed a framework where alternative test data could be used to reduce or replace traditional animal toxicity testing.

Trade Organization: Created a database and conducted hazard assessments for approximately 800 chemicals.

Town: Reviewed a cancer cluster analysis conducted by the state and communicated findings to the community.

University: Described the main features of internal dose time courses that are important when pharmacodynamics are governed by an activation threshold. Presented the adverse outcome pathway (AOP) for NLRP3-induced chronic inflammatory diseases as a case study.

Trade Organization: Evaluated the Consumer Product Safety Commission's Final Rule: Prohibition of Children's Toys and Child Care Articles Containing Specified Phthalates, including the methods used to calculate Hazard Index values.

Cleaning Products Company: Determined whether several ingredients of several cleaning products could have caused or exacerbated several claimed health effects (such as respiratory effects) in individuals using the products or working in areas where the products were used.

Public School: Evaluated school facility information and environmental assessments that have been conducted on lead in school drinking water. Quantified typical student exposures and estimated health risks. Evaluated exposures and risks of PEX replacement pipes.

Research Organization: Estimated dietary phthalate intake based on data from the National Health and Nutrition Examination Survey (NHANES), a program of studies of United States residents conducted at the Centers for Disease Control and Prevention (CDC).

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Trade Association: Reviewed and provided comments to the United States Environmental Protection Agency (US EPA) on its assessment plan for an updated arsenic health risk assessment.

Trade Organization: Developed a framework for a systematic, objective, and transparent evaluation of the evidence for non-cancer causation that requires reviewers to conduct a systematic study quality analysis and consider how the evidence from several realms impacts the interpretation of others.

Electric Utility: Evaluated the potential non-cancer and cancer health effects from exposure to coal combustion residuals (CCR) as a whole, and arsenic and chromium specifically, as reported in epidemiology and toxicity studies. Determined whether potential exposures to CCR contributed to current or future health effects, or warrant medical monitoring.

Trade Organization: Determined the safety of benzoic acid and its salts when used as preservatives in food and soft drinks based on an evaluation of pharmacokinetic data in rodents and humans, and human clinical studies of sodium benzoate administered as a therapeutic drug.

Children's Personal Care Product Manufacturer: Conducted a comprehensive hazard and risk assessment using data reported by various research and regulatory agencies, and specific risk assessments for individual preservatives, to determine whether there could be health risks for children from regular use of personal care products containing these preservatives.

Trade Organization: Evaluated the association between coffee generally, and acrylamide specifically, and cancer risk in the context of California's Proposition 65.

Trade Association: Conducted two comprehensive critical weight-of-evidence (WoE) reviews of studies bearing on the ability of very low bisphenol a (BPA) exposures to affect reproduction and development *via* endocrine disruption. These analyses were presented to several state legislative committees, all of which were considering bans on BPA.

Trade Organization: Conducted a survey of nearly 50 WoE frameworks to evaluate best practices for determining causation. Defined the key concepts of WoE analyses and their application to particular problems, and articulated the best practices from among the spectrum of approaches.

Food Manufacturer: Evaluated the significance of lead in imported hot sauces after a journal article reported that some of the products contained elevated levels of lead. Prepared a critique and summary of the study findings, and compared lead levels to US FDA limits. Evaluated the potential impact of the lead exposures on blood lead levels.

Trade Organization: Evaluated potential health risks from BPA in epoxy-lined metal cans based on both peer-reviewed scientific literature and regulatory agency risk assessments.

Personal Care Product Company: Conducted a risk assessment of zinc oxide in sunblock.

Trade Association: Critically evaluated the Environmental Benefits Mapping and Analysis Program – Community Edition (BenMAP-CE) that US EPA uses in the risk assessments for ozone (O<sub>3</sub>) and particulate matter (PM) as part of its NAAQS evaluation.

Chemical Company: Evaluated the utility of using epidemiology data in human health risk assessment and regulatory decision-making for the insecticide, chlorpyrifos.

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Trade Organization: Performed an O<sub>3</sub> mortality risk assessment using US EPA's Environmental Benefits Mapping and Analysis Program (BenMAP). Evaluated mortality risks by conducting a series of sensitivity analyses to assess how alternative model inputs impacted risk results.

Hospitals: Conducted screening level risk assessment for contaminants, including hexavalent chromium and polycyclic aromatic hydrocarbons, in product residue on surgical instruments used for medical procedures at several hospitals.

Industrial Consortium: Contributed to a toxicity and risk assessment in a class-action lawsuit by residents claiming adverse health effects from TCE and PCE in groundwater. Participated in a quantitative analysis of ingestion exposure, showering exposure, potential health risks, and proposed medical monitoring.

Water Supply Company: Evaluated potential health effects of arsenic, lead, and chlorination disinfection byproducts in drinking water.

City: Evaluated whether a career as a firefighter is associated with brain or lung cancer.

Consumer Product Company: Reviewed the safety testing required for a pesticide to be registered in the US; the potential risks and benefits of DEET; and standards, guidelines, and recommendations for using DEET.

Law firm: Evaluated whether air pollution may have increased the incidence and prevalence of several health conditions, including several cancers, in a city in Israel.

Trade Organization: Evaluated whether there is a scientific consensus regarding the potential health effects of asbestos compared to other elongate mineral particles, and whether any differences should be considered for testing guidelines.

Consumer Product Company: Critically reviewed epidemiology studies of specific consumer products.

Mining Company: Assessed the potential health risks of residents exposed to nickel as result of residing near a surface lateritic nickel mine and ferronickel smelter based on air, water, soil, and sediment data collected as part of the mine's environmental monitoring program.

Trade Association: Conducted a systematic review ozone exposure and metabolic syndrome.

Trade Organization: Critically reviewed parabens and weight gain epidemiology, toxicology, and mode-of-action evidence.

Waste Disposal Company: Evaluated the scientific evidence regarding radiation exposure and renal cell carcinoma in general and the likelihood that this cancer could have been caused by exposure to radionuclides from living in proximity to a landfill containing radioactive waste.

Trade Organization: Reviewed a cancer cluster investigation and epidemiology studies of pediatric leukemia and lymphoma, pediatric brain cancer, and pediatric Ewing sarcoma.

Trade Organization: Critically reviewed epidemiology research on air pollution and COVID.

Trade Organization: Evaluated and provided comments on US EPA's draft risk evaluation for trichloroethylene. Focused on the meta-analyses of kidney cancer, liver cancer, and non-Hodgkin's lymphoma.

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Non-Profit Research Institute: Developed an *in silico*-based health-protective screening approach for inhaled chemicals.

Law firm: Evaluated the potential human carcinogenicity of formaldehyde and methyl *tert*-butyl ether (MTBE).

Trade Organization: Conducted a systematic review of long-term exposure to fine particulate matter (PM<sub>2.5</sub>) and all-cause mortality.

Waste Management Company: Evaluated whether specific health conditions, including cancer, were likely attributable to exposures to Radium 226, Thorium 230, or Uranium 238 that originated from a landfill.

Farm: Evaluated whether certain health conditions could be caused by exposures to nitrate in drinking water or hydrogen sulfide or ammonia in air and, if so, under what exposure conditions.

Trade Organization: Conducted a WoE analysis of talc and ovarian cancer, including a quantitative bias analysis of epidemiology studies.

Trade Organization: Evaluated the association between personal PM<sub>2.5</sub> exposures and ambient PM<sub>2.5</sub> concentrations, and the implications for the interpretation of epidemiology studies that estimate personal exposure based on ambient concentrations.

Private Company: Assessed whether appropriate epidemiology methods were used to evaluate a potential pediatric cancer cluster in a military housing complex. Evaluated whether public health and environmental investigations used methodologically sound analyses.

Consumer Product Company: Determined whether a framework for assessing the hazard of cleaning product ingredients was sufficient to support "non-toxic" claims on product packaging for a household cleaner.

Consumer Product Company: Evaluated the historical state of knowledge regarding the toxicity of butadiene and the development of myelodysplastic syndrome.

Railroad Company: Summarized the historical states of knowledge regarding the toxicity of vermiculite and asbestos in the railroad industry.

Trade Organization: Conducted a systematic review of metallic nickel and cancer.

Trade Organization: Evaluated best practices for evidence integration in National Ambient Air Quality Standards (NAAQS) Integrated Science Assessments (ISAs).

State Environmental Agency: Quantitatively evaluated how uncertainty and bias can impact epidemiology associations between air pollutants and respiratory morbidity at low exposures.

Law Firm: Evaluated the potential lung cancer, mesothelioma, and interstitial fibrosis risks from exposure to chrysotile asbestos from brakes based on epidemiology studies of vehicle brake repair workers and industrial hygiene, mode-of-action, and toxicology data.

Chemical Company: Evaluated and provided comments on US EPA's Toxicological Review of Libby Amphibole Asbestos. Interacted with several regulatory agencies throughout the interagency process of the Integrated Risk Information System (IRIS) review.

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Trade Organization: Evaluated systematic review methods used by US EPA in IRIS and Toxic Substances Control Act (TSCA) evaluations.

Pesticide Companies: Evaluated the use of neurodevelopmental epidemiology studies by the US EPA in the re-registration process for organophosphate pesticides.

Consumer Product Company: Determined whether aspiration of a laundry pod caused long-term health effects in an infant.

Consumer Product Company: Evaluated the implications of a new toxicity study of an ingredient in a consumer product for adults and children who use the product and workers who manufacture the product. Provided an analysis of the potential Globally Harmonized System of Classification and Labeling of Chemicals (GHS) and Safety Data Sheet (SDS) implications of a the ingredient.

Trade Organization: Assessed whether a post-market skin patch epidemiology study should be used for risk assessment.

Trade Organization: Evaluated emission limits for several chemicals defined in certification guidelines for a consumer product.

Trade Organization: Reviewed and provided comments on Health Canada's Draft Screening Assessment Report and Risk Management Scope for talc.

Municipality: Evaluated whether childhood blood lead levels in a city appeared to be impacted by increases in water lead levels. Assessed the potential beneficial impact of the City's distribution of point-of-use water filters to residents.

Trade Organization: Developed detailed criteria for exposure characterization that can be used when designing future epidemiology studies and for evaluating completed studies to determine how their results should be considered in a regulatory setting.

Trade Organization: Determined whether nickel should be classified as a reproductive or developmental toxicant under California EPA's Proposition 65.

Consumer Product Company: Developed a statistical approach for a clinical study of a medical device.

Trade Organization: Performed a detailed quantitative analysis to determine the reliability and adequacy of the T25 Carcinogenic Potency Method for inhalation exposures of inorganic substances. This method is used to classify carcinogenic substances according to the European Union's guidance for Classification, Labelling and Packaging of Substances and Mixtures.

Law Firm: Evaluated the potential side effects and dose-response relationships for cosmetic botulinum toxin injections from reviews of clinical trials and FDA warning labels. Assessed whether claimed health effects in an individual existed prior to an injection with botulinum toxin, were included among the documented side effects of the toxin, or were indicative of systemic toxicity.

Trade Organization: Critically reviewed the draft recommendation for a non-health-based BPA occupational exposure limit (OEL) proposed by the Dutch Expert Committee on Occupational Health and Safety (DECOS). Derived and recommended a health-based BPA OEL that is consistent with European Commission directives.

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Law Firm: Evaluated nickel concentrations in an urban neighborhood using air monitoring data. Assessed the cancer and non-cancer health risks of exposure to nickel in air and dust among residents.

Trade Organization: Evaluated the association between short-term exposures to PM<sub>2.5</sub> and hospital admissions for cardiovascular diseases.

Research Organization: Evaluated the impact of respiratory infections, outdoor pollen, and socioeconomic status on associations between PM<sub>2.5</sub> and ozone and pediatric asthma hospital admissions.

Research Organization: Critically reviewed the epidemiology literature on exposure to PM<sub>2.5</sub> and several birth outcomes.

Trade Organization: Reviewed and commented on the International Agency for Research on Cancer (IARC) Preamble, which summarizes the underlying scientific principles of the IARC Monographs, which evaluate the carcinogenic hazards of chemicals and other substances.

Baby Product Company: Evaluated whether a teething ring product contained more Bisphenol A than is permitted under Proposition 65.

Energy Company: Evaluated whether working on the site of a former Manufactured Gas Plant may have contributed to kidney cancer.

Steel Company: Assessed the state of knowledge related to employment in a steel mill or steel plant and asbestos-related disease to understand when the scientific community began to study the hazards of asbestos among steelworkers and when it was reasonable for a steel manufacturing company to have known that exposure to asbestos-containing materials could potentially lead to the development of respiratory disease in steelworkers.

Trade Organization: Evaluated US EPA's calculation of an inhalation unit risk (IUR) in its "Toxicological Review for Trichloroethylene in Support of Summary Information on the IRIS."

Trade Organization: Developed standards for epidemiology studies similar to good laboratory practice (GLP) standards, which are intended to assure the quality and integrity of non-clinical laboratory studies.

Manufacturer: Critically reviewed the quality of case-control studies conducted in North America that assessed the associations between captan exposure and the risk of multiple myeloma. Conducted a quantitative bias/uncertainty analysis of these studies.

Trade Organization: Evaluated the basis for the American Conference of Governmental Industrial Hygienists (ACGIH) lowering the Threshold Limit Value for toluene diisocyanate.

Law Firm: Evaluated exposure to O-toluidine and bladder cancer risk.

Law Firm: Evaluated exposures to landfill gases, including hydrogen sulfide, and potential health effects from these exposures in individuals residing near a municipal solid waste landfill. Evaluated potential odor impacts and the differences between odor perception and adverse health effects.

Trade Organization: Reviewed literature regarding several air toxics and health endpoints and provided recommendations on a proposed regulation in California to monitor communities for air pollution-attributable health effects.

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Trade Organization: Critically reviewed the harmonized carcinogenicity classification and labeling for cobalt metal developed to comply with the Registration, Evaluation, Authorisation and Restriction of Chemicals (REACH) regulation by RIVM (Netherlands National Institute for Public Health and the Environment).

Toy Manufacturer: Evaluated whether brain injury could occur as a primary consequence of ingesting toy beads containing 1,4-butadiene, or as a secondary consequence of experiencing coma-like symptoms following ingestion of the beads.

Pharmaceutical Company: Assessed whether on-label use of a pharmaceutical increased cardiovascular disease risk based on randomized controlled trials and observational epidemiology studies.

US EPA: Contributed to the design of a model describing several frequently encountered toxicity endpoints in terms of a series of progressive pathophysiological steps.

Pharmaceutical Company: Performed an in-depth analysis of the toxicology and epidemiology data of a specific drug to determine whether the company could have anticipated potential adverse side effects in humans.

Pharmaceutical Company: In the context of a patent infringement lawsuit, performed an independent analysis of efficacy and toxicity data from animal experiments to determine if claims in the patent could be challenged.

Government Agency: Critically evaluated an epidemiology study of exposures and health outcomes in indigenous and Afro-Colombian communities living near a nickel mine and smelter and discussed with the government agency that conducted the study.

Chemical Company: Conducted a meta-analysis of epidemiology studies that evaluated the use of a pharmaceutical during labor and dystocia.

Trade Association: Evaluated whether atherosclerosis development is a plausible mode of action for PM in cardiovascular pathogenesis, and whether this is supported by epidemiology evidence.

Trade Association: Critically reviewed the draft recommendation for a non-health-based OEL for di- and triisocyanates proposed by the DECOS of the Health Council of the Netherlands.

Trade Organization: Developed a database of epidemiology studies of occupational exposures to pesticides and cancer.

Trade Organization: Critically reviewed the epidemiology literature on long-term exposure to ambient ozone and asthma development.

Manufacturer: Evaluated the health risks of potential carbon monoxide exposures at a proposed bridge near a manufacturing facility.

Consumer Product Company: Systematically reviewed epidemiology, toxicity, exposure and transport, and mechanistic studies to evaluate whether personal use of cosmetic talc increases ovarian cancer risk.

Trade Organization: Evaluated whether an alternative form of the sulfur dioxide (SO<sub>2</sub>) NAAQS would be as public health protective as the then-current form.

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Trade Organization: Critically reviewed the approach US EPA used to quantify health co-benefits when assessing the impacts of the Clean Power Plan. Evaluated the scientific validity of the models, data sources, and assumptions underlying US EPA's calculations.

Chemical Company: Evaluated whether exposure to odorous chemicals emitted from spray foam insulation in a residence posed a potential health risk. Compared residents' exposure to odor thresholds, toxicity criteria, and health effect levels for specific constituents emitted from the foam.

Law Firm: Assessed human health risks associated with coal-fired power plant emissions, including particulate matter, SO<sub>2</sub>, and nitrogen oxides (NO<sub>x</sub>), based on air modeling results and available measurement data.

Trade Organization: Conducted a systematic review of epidemiology studies that evaluated proximity to unconventional natural gas development (UNGD) and perinatal outcomes.

Chemical Company: Provided chemical-level assessments (*i.e.*, analysis of inventory status, occupational exposure limits, and available toxicity data) and product-level assessments (*i.e.*, evaluation of Good Manufacturing Practice and regulatory hurdles) for several cosmetics and consumer products.

Research Organization: Critically evaluated toxicity studies that investigated PM<sub>2.5</sub> and developmental and reproductive effects.

Utility Group: Evaluated potential exposures and health impacts of PM in general, as well as two specific types of PM associated with power plant operations (*i.e.*, diesel exhaust emissions and coal dust emissions).

Manufacturer: Assessed the potential health risks of saline-filled breast implants based on a review of the peer-reviewed literature and pre- and post-market studies of silicone- and saline-filled breast implants.

Pharmaceutical Company: Evaluated whether historical data support the patentability of a drug that lowers blood lipid levels. Reviewed the experimental methods (including statistics) and results of efficacy studies with the drug to confirm the original study conclusions.

Trade Associations: Facilitated research that addresses the causality of the relationship between PM<sub>2.5</sub> and mortality. Selected research candidates, developed a request for proposal, evaluated solicited proposals, coordinated selected researchers for data access, and organized a symposium for researchers to present and discuss their findings.

Utility Company: Evaluated health effects associated with potential exposures to chemicals, including coal tar and polycyclic aromatic hydrocarbons (PAHs) such as benzo[a]pyrene, resulting from recreational activities in the vicinity of a former manufactured gas plant site.

Chemical Company: Evaluated whether the PAHs in an asphalt roofing underlayment would require its classification under the Occupational Safety and Hazard Administration's (OSHA) hazard communication standard.

Manufacturer: Evaluated the incidence, prevalence, risk factors, and potential alternative causes of health complaints among office workers allegedly exposed to chemicals in indoor air *via* vapor intrusion of contaminated groundwater. Also assessed whether there were any disease clusters at the site.

Trade Organization: Provided written and oral comments to the Clean Air Scientific Advisory Committee (CASAC) on exposure, epidemiology, toxicity, and mode-of-action studies and their bearing on US EPA's development of NAAQS for O<sub>3</sub>, PM, NO<sub>x</sub>, and sulfur oxides (SO<sub>x</sub>) on numerous occasions.

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Manufacturer: Reviewed asbestos exposure data related to the handling and installation of electrical equipment and epidemiology evidence regarding mesothelioma, lung cancer, and laryngeal cancer in electricians.

US Army: Assessed several dose-response functions, animal models, and endpoints to obtain the most appropriate model-predicted risk estimate to use in establishing a risk-based inhalation exposure criterion for vanadium pentoxide.

Appliance Manufacturer: Evaluated evidence for a cancer cluster in a neighborhood where soil containing polychlorinated biphenyl (PCBs) was historically used as landfill in a recreation area.

State Environmental Agency: Conducted WoE evaluations of the association between short-term and long-term ozone exposure and cardiovascular effects.

State Environmental Agency: Evaluated how meta-analyses have been or could be applied in the evaluation of health effects of air pollutants. Assessed the strengths and limitations of methods, issues that arise in meta-analyzing different types of data, assumptions that can influence the interpretation of results, and how bias and heterogeneity can be addressed.

Law Firm: Assessed the impact of coal-fired power plant emissions on exposures to fine PM, SO<sub>2</sub>, and NO<sub>2</sub> and whether these exposures likely contribute to adverse health effects in Colorado residents.

Mining Company: Evaluated methods used to derive a proposed standard for nickel by the Province of Quebec, Canada.

Utility Company: Reviewed the historical releases of compounds associated with manufactured gas plant processes, including PAHs such as benzo[a]pyrene, and the historical understanding of risk assessment, carcinogenesis, and the toxicity of these compounds.

State Environmental Agency: Reviewed epidemiology, controlled human exposure, experimental animal, and mechanistic studies of ozone and markers of inflammation and oxidative stress.

State Environmental Agency: Critically reviewed potential uses of Next Generation (NexGen) toxicity testing methods and associated interpretation techniques in assessing the risks associated with chemical exposures (e.g., in high-throughput screening programs or detailed quantitative analyses for individual data-rich chemicals).

Trade Organization: Critiqued draft templates for tabulating epidemiology and experimental animal study data for hazard identification proposed by the Developmental and Reproductive Toxicant Identification Committee (DART IC) of California's Office of Environmental Health Hazard Assessment (CalOEHHA). Proposed an alternative set of tables to systematically present data for consideration in a full evidence integration process.

State Environmental Agency: Critically reviewed epidemiology, controlled human exposure, experimental animal, and mechanistic studies of ozone and outcomes related to asthma exacerbation.

State Environmental Agency: Evaluated the relationship between ozone concentrations and asthma hospitalizations in Texas from 2001-2011.

Trade Organization: Conducted sensitivity analyses to determine how alternative assumptions impact exposure and risk estimates calculated using the US EPA Air Pollutants Exposure (APEX) Model.

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Health Care Company: Analyzed the health effects of perchloroethylene and its breakdown products (trichloroethylene, 1,1-dichloroethylene, 1,2-dichloroethylene, and vinyl chloride) to address community concerns with remediation requirements. Also reviewed the bases for soil and groundwater remediation goals.

Chemical Company: Analyzed health risks from exposure to PCBs in caulking in a school in Massachusetts by analyzing potential exposures at the school and epidemiology and toxicology evidence. Also summarized the state of knowledge regarding PCBs when the school was built.

State Environmental Agency: Organized and participated in a workshop focused on the scientific evidence for ozone effects and the societal implications of lowering the ozone NAAQS.

Utility Company: Characterized and communicated exposures to chemicals, including PAHs such as benzo[a]pyrene, for occupants of residential and commercial properties constructed on the site of a former manufactured gas plant.

Trade Association: Evaluated non-cancer effects of PM.

Chemical Company: Evaluated whether inflammation and oxidative stress caused by ozone exposure are key events leading to respiratory toxicity.

Chemical Company: Conducted a WoE evaluation of PM and biomarkers of cancer.

Chemical Company: Evaluated whether there was evidence of any developmental health effects clusters at an office building. Assessed likely explanations for health claims.

Trade Association: Proposed a framework for evaluating causation that provides a structure for integrating different realms of evidence, weighing the strength of evidence for causation, and assessing the potential impact of uncertainty on the body of evidence.

Trade Organization: Critically reviewed the epidemiology literature evaluating nickel exposure and reproductive and developmental effects. Determined whether it supported a prioritization of nickel for CalOEHHA's consideration under California's Proposition 65.

Trade Organization: Evaluated whether higher regulatory limits on heavy metals in finished medical marijuana products would provide an adequate margin of safety for patients, and whether certain hydrocarbon gases could be safely used in the production of cannabis concentrates used in medical marijuana infused products.

Law Firm: Evaluated whether exposures to diesel exhaust and jet fuel are associated with lung cancer based on a literature review, agency classifications, and key epidemiology studies on which classifications are based.

Electric Utility: Evaluated the scientific basis of health impacts associated with air quality regulations that would impact an electricity generation facility. Compared air quality data in the area around the facility to health-based NAAQS.

Trade Organization: Assessed the US EPA "Framework for Human Health Risk Assessment to Inform Decision Making" and compared it to US EPA's ongoing O<sub>3</sub> analysis. Focused on planning and "fit for purpose," WoE, transparency, reasonableness, consistency, at-risk factors, and uncertainty and variability.

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Electric Utility: Analyzed air monitoring data to determine the potential public health impacts of stack air emissions of fine PM, O<sub>3</sub>, NO<sub>2</sub>, and SO<sub>2</sub>.

Trade Organization: Conducted a quantitative analysis of controlled human exposure studies to address whether there is a subset of individuals who are susceptible to health effects of criteria air pollutants at particular exposure levels, but whose response is obscured by analyzing data at the group level.

Law Firm: Analyzed human health risks posed by chemicals measured in workplace indoor air that were alleged to have originated from groundwater contaminated by a nearby recycling facility. Focused on the epidemiology of the chemicals of concern at the levels measured in the workplace and the plaintiffs' health complaints, including cancer.

Smelter: Assessed whether a smelter's permit would likely allow for SO<sub>2</sub> emissions that could lead to adverse health effects in the community.

Trade Organization: Reviewed the basis for the California Environmental Protection Agency's (CalEPA's) proposal to list SO<sub>2</sub> as a Proposition 65 developmental and reproductive toxicant. Evaluated whether the underlying studies provided sufficient and robust evidence that SO<sub>2</sub> causes developmental and reproductive effects.

Trade Organization: Reviewed and critiqued the assumptions and uncertainties associated with the statistical models on which US EPA's 2011 Benefits and Costs of the Clean Air Act Report was based.

Chemical Company: Evaluated US EPA's proposed national emission standards for hazardous air pollutants (NESHAPs) for mercury from major industrial boilers. Evaluated the agency's statistical approach for establishing the maximum achievable control technology (MACT) limit and determined how alternative approaches would impact the MACT derivation.

Trade Organization: Conducted meta-analyses and meta-regressions of airway hyper-responsiveness in asthmatic volunteers exposed to NO<sub>2</sub> in clinical studies. Presented these analyses to the US Office of Management and Budget.

Trade Organization: Assessed what constitutes an adverse health effect vs. normal biological variation (or adaption or compensation to stressors), and the role of statistics in assessing adversity. Used airway hyper-responsiveness to SO<sub>2</sub> as a case study.

Health Effects Institute: Compiled and reviewed studies regarding chronic and acute toxicity guidelines for mobile source air toxicants.

Trade Organization: Evaluated studies examining low-dose exposure to BPA and effects on reproduction and development using a W approach. Discussed results in written comments and oral testimony to CalEPA in the context of whether BPA should be listed as a female reproductive toxicant under Proposition 65.

Cleaning Product Company: Evaluated toxicity of chemicals in all-natural cleaning products.

Municipality: In response to citizens' concerns, independently investigated whether there was an increased incidence of cancer in residents living near a municipal landfill. Communicated findings with city officials and residents at public meetings.

Toy Distributor: Determined whether a toxicological evaluation of a toy was sufficient for determining children's health risks. Assessed the toxicity of a chemical found in the toy, potential routes of exposure, and possible health risks.

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Trade Organization: Conducted a systematic review and meta-analyses of 2,4-dichlorophenoxyacetic acid (2,4-D) and non-Hodgkin's lymphoma, gastric cancer, and prostate cancer. Participated as an observer at the IARC Monograph 113 Meeting at which 2,4-D was evaluated.

Trade Organization: Assessed whether animal, mechanistic, and epidemiology evidence is consistent with the nickel ion bioavailability model, which asserts that the carcinogenicity of nickel-containing substances is based on the bioavailability of the nickel ion at nuclear sites of target respiratory epithelial cells.

Public Agency: Evaluated the variability in water lead levels across a US city and the association between water lead levels and blood lead levels in children.

Pesticide Company: Assessed whether epidemiology, toxicology, and mechanistic evidence support chlorpyrifos being a neurobehavioral toxicant in humans at relatively low exposure levels. Evaluated evidence using recently proposed frameworks for integrating human and animal data, as well as Gradient's hypothesis-based weight-of-evidence (HBWoE) approach.

Cleaning Product Company: Evaluated the potential risks of birth defects from exposure to the chemical components of products used in floor stripping and refinishing.

Trade Organization: Conducted a critical examination of a proposal by the National Academy of Sciences that linear low-dose extrapolation should be used for non-cancer and cancer endpoints as a default because measurement error in epidemiology studies linearizes dose-response curves.

Law Firm: Reviewed specific exposure information and occupational epidemiology literature for a claim regarding a causal association between formaldehyde inhalation and acute myeloid leukemia.

Law Firm: Evaluated whether radiation should have been considered as a potential cause of an individual's mesothelioma. Analyzed both specific exposure information and toxicology and epidemiology literature on radiation and mesothelioma.

Trade Association: Using the HBWoE approach, evaluated whether epidemiology, toxicology, and mechanistic evidence supports the plausibility of formaldehyde as a human leukemogen.

Chemical Companies: Calculated a benchmark dose (BMD) for an industrial chemical using US EPA's BMD Software (BMDS). Assessed several dose-response models and evaluated the impact of using historical control data.

Trade Organization: Evaluated whether epidemiology, animal toxicity, mechanistic, and pharmacokinetic evidence indicates that toluene diisocyanate is a human carcinogen.

Trade Organization: Critically reviewed meta-analysis of respiratory cancer risk following inhalation exposure to nickel compounds. Provided comments regarding the methods, limitations, and interpretation of results throughout the conduct of this study.

Trade Organization: Conducted a pilot meta-analysis of studies bearing on the ability of very low oral exposures to BPA to affect prostate weight in rodents. Investigated the possibility of publication bias and evidence for a temporal trend in the data.

Trade Organization: Critically reviewed a draft European Union report on the state of the science regarding endocrine-disrupting chemicals. Assembled a panel of experts to determine whether the draft report constituted a complete and unbiased analysis of endocrine disruptors.

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Trade Organization: Reviewed epidemiology studies assessing associations between BPA and several health effects.

Trade Association: Reviewed and prepared comments on ACGIH's proposed Threshold Limit Value for manganese. Reviewed the methodology applied by ACGIH, compared the use of published regression analyses of manganese dose-response data to benchmark dose modeling of more recent data, and identified appropriate adverse effect levels of manganese in occupational studies.

Chemical Manufacturer: Reviewed the epidemiology and mode-of-action data on acetic anhydride and cancer using a systematic WoE approach to determine whether the data are consistent with ACGIH cancer classification.

Law Firm: Evaluated epidemiology literature regarding present and future risks of cancer and non-cancer health effects in a group of individuals from inhalation exposures to trichloroethylene (TCE) and perchloroethylene (PCE).

Law Firm: Evaluated the epidemiology literature regarding cancer and non-cancer health effects of benzene, dioxin, and pentachlorophenol. Conducted a cluster analysis to determine whether individuals residing in an area with alleged exposures had increased rates of several cancers and non-cancer health effects.

Pesticide Companies: Critically reviewed epidemiology, toxicology, and mechanistic studies to assess whether exposure to atrazine in drinking water is associated with reproductive and developmental health effects.

Confidential Client: Evaluated the health effects associated with hexavalent chromium based on an assessment of the epidemiology literature. Assessed the scientific rigor of an analysis of potentially exposed individuals' survey responses.

Trade Organization: Classified, summarized, and entered relevant lead studies into the International Uniform Chemical Information Database (IUCLID) 5.2, a database for the intrinsic and hazard properties of chemical substances that companies can use to submit data under the REACH legislation in Europe.

Trade Organization: Provided written and oral testimony to the US National Toxicology Program (NTP) and its Board of Scientific Councilors regarding occupational epidemiology studies of styrene and whether styrene should be considered a human carcinogen.

Trade Organization: Developed scientifically sound approaches for incorporating human data into quantitative non-cancer risk assessment to support commentary on the ongoing US EPA revision of its dioxin assessment.

Trade Organization: Evaluated the associations between metal exposures and health outcomes using NHANES data.

Trade Organization: Conducted a WoE assessment of exposure to soluble nickel compounds and respiratory cancer risk based on animal carcinogenicity, mode-of-action, and occupational epidemiology studies.

Trade Organization: Conducted a critical review of the US EPA Toxicological Review of 1,4-Dioxane in support of "Summary Information" provided in IRIS. Proposed alternative methods to calculate the cancer slope factor and reference dose.

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Law Firm: Evaluated the toxicology and epidemiology evidence regarding several pesticides and whether there was evidence for a causal association with certain birth defects.

Law Firm: Assessed recent occupational epidemiology studies of manganese and their bearing on the reference concentration (RfC).

Law Firm: Assessed whether epidemiology literature supports an association between low-level exposures to lead and IQ.

Trade Organization: Determined whether linear low-dose extrapolation should be used for non-cancer endpoints.

Law Firm: Assessed appropriateness of statistical analyses used by the Ramazzini Foundation for comparing cancer incidence rates in rats treated with MTBE and untreated rats.

Law Firm: Critically reviewed epidemiology literature of radium and osteosarcoma risk. Determined whether osteosarcoma rates were higher than expected in certain geographic regions in a southern state.

Law Firm: Critically reviewed potential health effects associated with exposure to heating oil from a basement spill.

Law Firm: Critically reviewed the epidemiology literature on the role of ionizing radiation in cancer risk in patients receiving radiation therapy, in nuclear energy facility workers, and in patients receiving Thorotrast treatments.

Chemical Company: Conducted a comprehensive review of the scientific literature on indoor dust levels of several flame retardants and an exposure assessment of each one.

Consumer Product Company: Interpreted the results of two genotoxicity screening assays in light of their sensitivity and specificity.

Toy Manufacturer: Conducted failure analyses of children's toys to determine whether proper or improper use was likely to lead to physical harm. Made recommendations regarding ways to make the toys safer.

Chemical Manufacturing Plant: Evaluated the mercury toxicology and epidemiology literature and determined whether levels in residential soil were above background and likely attributable to a nearby manufacturing plant.

Chemical Company: Conducted a critical review of neurodevelopmental toxicity studies of the flame retardant, decabromodiphenyl ether.

Petroleum Refining Company: Conducted an uncertainty analysis of the carcinogenicity of naphthalene using an HBWoE approach.

Power Plant: Critically reviewed published epidemiology studies of health effects in children residing near coal-fired power plants or coal mines. These studies examined respiratory outcomes, birth defects, and effects on physical development.

Law Firm: Analyzed health effects – including fetal, infant, and total death rates and cancer rates – and certain vital statistics in a Montana county. Compared overall health status of the county to that of the state.

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Trade Organization: Evaluated and applied an uncertainty analysis focused on dioxin exposure and health effects data from key toxicology and human biomonitoring-based epidemiology studies as part of a margin-of-exposure analysis.

Law Firm: Evaluated toxicology and epidemiology evidence regarding glutaraldehyde and hydroquinone exposure and leukemia.

Trade Organization: Developed and refined a search strategy for the exposure and health effects of a chemical used in a manufacturing process using several databases, such as PubMed, Toxline, IRIS, and HSDB. Identified and screened relevant articles for inclusion in an electronic database.

Cleaning Product Company: Designed methodology for testing the presence and activity of an enzyme in a cleaning product. Determined whether this enzyme was appropriate for the product.

Wood Treatment Plant: Analyzed dioxin and PCB congeners in individuals residing near a wood treatment plant and compared them to background levels reported by NHANES. Analyzed these compounds in soil, dust, and sediment to determine whether there were elevated risks of exposure.

Small Business: Assessed whether cancer cases at a small business could be attributed to a common exposure.

Trade Association: Re-analyzed published rat testicular carcinogenicity data on MTBE using the Poly-3 statistical method to account for survival differences among treatment groups.

Pesticide Company: Analyzed US EPA's use of the lower confidence limit on the benchmark dose (BMDL<sub>10</sub>) to determine a point of departure for the cancer risk of dimethylarsenic acid in humans.

Research Organization: Critically reviewed epidemiology literature to determine if the effects of lead and mercury on human neurological development are additive or synergistic.

Flame Retardant Company: Provided toxicological, database, and risk analysis support for product development of phosphorus-based flame retardant chemicals with low potential for health and environmental impact.

Chemical Company: Contributed to the drafting of an evidence-based argument submitted to US EPA regarding whether acetonitrile should be delisted from the US EPA's Toxic Release Inventory.

Smelter: Reviewed general and company-specific historical knowledge of human and ecological toxicity of smelter contaminants.

Manufacturer: Summarized the cancer and non-cancer effects of cobalt and nickel for a company that fabricates tungsten heavy metal alloy products.

**Publications – Journal Articles**

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Goodman, JE; Yu, CJ. 2009. "Target sites: Skin." In *Information Resources in Toxicology (Fourth Edition)*. (Ed.: Wexler, P), Elsevier, Amsterdam, Netherlands, p481-484.

Goodman, JE; Rhomberg, LR. 2009. "Bisphenol A." In *Endocrine Disrupting Chemicals in Food*. (Ed.: Shaw, I), Woodhead Publishing, Cambridge, United Kingdom, p406-436.

**Julie E. Goodman, Ph.D., DABT, FACE, ATS****Publications – Abstracts and Posters**

Boon, DN; Khandaker, A; Annesar, EG; Goodman, JE. 2023. "Epidemiology Study Quality Evaluation in a Systematic Review of Aspartame and Cancer Evidence." Poster #LP-61. Presented at the EUROTOX Congress 2023, Ljubljana, Slovenia, September 10-13.

Ticknor, RC; Zhou, J; Goodman, JE. 2022. "Systematic Review of Perchloroethylene and Non-Hodgkin's Lymphoma." Poster # 3928/P607. Presented at the Society of Toxicology (SOT) 61<sup>st</sup> Annual Meeting & ToxEpo, held March 27-31.

Prueitt, RL; Li, W; Zhou, J; Goodman, JE. 2021. "Systematic Review of the Association Between Long-Term Exposure to Ambient Fine Particulate Matter and Mortality." Abstract/Poster: 2304/P180. Presented at the Society of Toxicology (SOT) 60<sup>th</sup> Annual Meeting & ToxEpo, held virtually March 21-26.

Prueitt, RL; Li, AW; Chang, RY; Goodman, JE. 2020. "Systematic Review of the Potential Respiratory Carcinogenicity of Metallic Nickel in Humans." Poster # 1516/P617. Prepared for presentation at the Society of Toxicology (SOT) 59<sup>th</sup> Annual Meeting & ToxEpo, Anaheim, CA, March 15-19 (Conference cancelled).

Boomhower, SR; Goodman, JE; Li, AW; Long, CM. 2020. "A Systematic Review and Analysis of Personal and Ambient PM<sub>2.5</sub> Measurements: Implications for Epidemiological Studies." Poster # 1135/P177. Prepared for presentation at the Society of Toxicology (SOT) 59<sup>th</sup> Annual Meeting & ToxEpo, Anaheim, CA, March 15-19 (Conference cancelled).

Goodman, JE; Li, AW. 2020. "Using Causal Methods to Assess Whether Reductions in PM<sub>2.5</sub> Result in Decreased Mortality." 5p. Prepared for presentation at the Health Effects Institute (HEI)'s 2020 Annual Conference, Boston, MA, April 5-7 (Conference cancelled).

Zu, K; Goodman, JE; Prueitt, RL. 2019. "Strengthening the Evaluation of Mechanistic Evidence Categorized by the IARC 10 Key Characteristics of Carcinogens." Presented at the National Academies of Sciences, Engineering, and Medicine (NASEM) Evidence Integration Workshop, Washington, DC, June 3-4.

Goodman, JE; Johnson, G; Prueitt, RL; Zu, K. 2019. "Systematically Evaluating and Integrating Evidence in National Ambient Air Quality Standards (NAAQS) Reviews." Presented at the National Academies of Sciences, Engineering, and Medicine (NASEM) Evidence Integration Workshop, Washington, DC, June 3-4.

Zu, K; Goodman, JE; Prueitt, RL. 2019. "Evaluating Mechanistic Evidence: Beyond the IARC 10 Key Characteristic Framework for Carcinogens." Presented at the National Toxicology Program (NTP) Workshop: Converging on Cancer, Washington, DC, April 29-30.

Goodman, JE; Johnson, G; Prueitt, RL; Zu, K. 2019. "Systematically Evaluating and Integrating Evidence on Cancer in National Ambient Air Quality Standards (NAAQS) Reviews." Presented at the National Toxicology Program (NTP) Workshop: Converging on Cancer, Washington, DC, April 29-30.

Goodman, JE; Pizzurro, DM; Lemay, JC; Lewandowski, T; Zu, K. 2018. "Use of Chemical-specific Adjustment Factors in Health Risk Assessments: A Tale of Two Preservatives." Poster #175/OP02-04. Presented at EUROTOX 2018, Brussels, Belgium, September 2-5.

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Prueitt, RL; Lynch, HN; Mohar, I; Goodman, JE. 2018. "Critical Evaluation of Threshold for Respiratory Effects of Toluene Diisocyanate." Poster #701/P19-27. Presented at EUROTOX 2018, Brussels, Belgium, September 2-5.

Goodman, JE; Peterson, MK; Hixon, ML; Pacheco Shubin, S. 2018. "Derivation of a Maximum Allowable Dose Level for Bisphenol A." Poster #2903/P429. Presented at the Society of Toxicology (SOT) 57<sup>th</sup> Annual Meeting, San Antonio, TX, March 11-15.

Lynch, HN; Goodman, JE; Prueitt, RL; Mohar, I. 2018. "Critical Evaluation of Thresholds for Respiratory Effects of Toluene Diisocyanate." Poster #1197/P248. Presented at the Society of Toxicology (SOT) 57<sup>th</sup> Annual Meeting, San Antonio, TX, March 11-15.

Prueitt, RL; Lynch, HN; Zu, K; Shi, L; Goodman, JE. 2018. "Evaluation of Respiratory Cancer Risk from Dermal Exposure to Toluene Diisocyanate." Poster #2820/P343. Presented at the Society of Toxicology (SOT) 57<sup>th</sup> Annual Meeting, San Antonio, TX, March 11-15.

Liu, X; Zu, K; Lam, T; Goodman, JE. 2018. "Systematic Review and Meta-analysis of Diazepam and Labor Duration." Poster #1876/P207. Presented at the Society of Toxicology (SOT) 57<sup>th</sup> Annual Meeting, San Antonio, TX, March 11-15.

Zu, K; Goodman, JE; Pizzurro, DM; Lewandowski, TA. 2018. "Use of Clinical Data to Inform Risk Assessments of Food Additives: A Case Study of Sodium Benzoate." Poster #2847/P370. Presented at the Society of Toxicology (SOT) 57<sup>th</sup> Annual Meeting, San Antonio, TX, March 11-15.

Goodman, JE; Lynch, HN; Prueitt, RL; Mohar, I. 2017. "Evaluation of ACGIH TLVs for Toluene Diisocyanate." Poster #P.178. Presented at the Society for Risk Analysis (SRA) Annual Meeting, Arlington, VA, December 10-14.

Pizzurro, DM; Zu, K; Lewandowski, TA; Goodman, JE. 2017. "Use of Pharmacokinetic Data for Adjustment Factor Derivation for Benzoate Preservatives." Presented at American College of Toxicology (ACT) 38<sup>th</sup> Annual Meeting, Palm Springs, CA, November 5-8.

Zu, K; Liu, X; Shi, L; Tao, G; Loftus, C; Lange, S; Goodman, JE. 2017. "Concentration-response of Short-term Ozone Exposure and Hospital Admissions for Asthma in Texas." Presented at the Society of Epidemiological Research 50<sup>th</sup> Annual Meeting, Seattle, WA, June 20-23.

Lynch, HN; Goodman, JE. 2017. "Risk Assessment Calls for Standard Guidelines for Exposure Characterization in Epidemiology Studies." Presented at the Society of Epidemiological Research 50<sup>th</sup> Annual Meeting, Seattle, WA, June 20-23.

Lam, T; Loftus, C; Zu, K; Kennedy, EM; Goodman, JE. 2017. "2,4-Dichlorophenoxyacetic Acid and Soft Tissue Sarcoma: Meta-analysis of the Published Literature." Presented at the Society of Epidemiological Research 50<sup>th</sup> Annual Meeting, Seattle, WA, June 20-23.

Prueitt, RL; Goodman, JE. 2017. "Mode-of-action Evaluation for Ozone-induced Respiratory Effects Through Activation of Neural Reflexes." Poster #2824/P226. Presented at the Society of Toxicology (SOT) 56<sup>th</sup> Annual Meeting, Baltimore, MD, March 12-16.

Lynch, HN; Goodman, JE; Yan, Z. 2017. "Risk Assessment Calls for Standard Guidelines for Exposure Characterization in Epidemiology Studies." Poster #1311/P534. Presented at the Society of Toxicology (SOT) 56<sup>th</sup> Annual Meeting, Baltimore, MD, March 12-16.

**Julie E. Goodman, Ph.D., DABT, FACE, ATS**

Zu, K; Liu, X; Goodman, JE. 2017. "Meta-analysis of Dose-response Relationships from Epidemiology Studies for Risk Assessment: A Case Study of Trans Fats and Coronary Heart Disease." Poster #1850/P424. Presented at the Society of Toxicology (SOT) 56<sup>th</sup> Annual Meeting, Baltimore, MD, March 12-16.

Lemay, JC; Zu, K; Zhang, J; Goodman, JE. 2016. "Sunscreen Safety for Kids: A Risk Assessment of a Common Preservative." Poster #MP168. Presented at the Society of Environmental Toxicology and Chemistry (SETAC) North America 37<sup>th</sup> Annual Meeting, Orlando, FL, November 6-10.

Bailey, LA; Kerper, LE; Goodman, JE. 2016. "Occupational Exposure Level for Manganese in Welding Fumes Based on the Best Available Science." Presented at the Manganese 2016 Conference: Manganese Health Effects on Neurodevelopment & Neurodegenerative Diseases, New York, NY, September 25-28.

Goodman, JE; Zu, K; Tao, G; Rhomberg, LR. 2016. "Evaluation of Occupational Benzene Exposure and Leukemia Mortality: A Lifetable Analysis of the Pliofilm Cohort." Presented at the Epidemiology Congress of the Americas 2016 Annual Meeting, Miami, FL, June 21-24.

Zu, K; Tao, G; Goodman, J. 2016. "Pleural plaques and lung function in the Marysville worker cohort: A re-analysis." Presented at the Epidemiology Congress of the Americas 2016 Annual Meeting, Miami, FL, June 21-24.

Lange, SS; Tao, G; Rhomberg, LR; Goodman, JE; Dourson, ML; Honeycutt, ME. 2016. "Dose Response Curves Derived from Clinical Ozone Exposures Can Inform Public Policy." Presented at the Society of Toxicology (SOT) 55<sup>th</sup> Annual Meeting, New Orleans, LA, March 13-17.

Zu, K; Lemay, JC; Zhang, J; Goodman, JE. 2016. "A Hazard and Risk Assessment of Phenoxyethanol in Children's Personal Care Products." Presented at the Society of Toxicology (SOT) 55<sup>th</sup> Annual Meeting, New Orleans, LA, March 13-17.

Prueitt, RL; Cohen, JM; Goodman, JE. 2016. "Evaluation of Atherosclerosis as a Mode of Action for the Cardiovascular Effects of Particulate Matter." Presented at the Society of Toxicology (SOT) 55<sup>th</sup> Annual Meeting, New Orleans, LA, March 13-17.

Seeley, MR; Goodman, JE. 2016. "Evaluation of Nitrogen Dioxide and Airway Hyper-responsiveness." Presented at the Society of Toxicology (SOT) 55<sup>th</sup> Annual Meeting, New Orleans, LA, March 13-17.

Zu, K; Loftus, CT; Goodman, JE. 2015. "Non-Hodgkin's Lymphoma and 2,4-dichlorophenoxyacetic Acid: A Meta-analysis." Poster No. 022. Presented at the Society for Epidemiological Research 48<sup>th</sup> Annual Meeting, Denver, Colorado, June 19, 2015.

Rhomberg, LR; Mayfield, DB; Goodman, JE; Butler, EL; Nascarella, MA; Williams, DR. 2015. "Quantitative Cancer Risk Assessment for Occupational Exposures to Asphalt Fumes During Built-up Roofing Asphalt (BURA) Operations." Presented at the INRS 2015 Occupational Health Research Conference, Nancy, France, April 8-10.

Tao, G; Zu, K; Long, CM; Goodman, JE; Valberg, PA. 2015. "Forest-fire Fine Particulate Matter and Daily Mortality in Greater Boston and New York City." *Toxicologist* 144(1):163. Abstract 763. Presented at the Society of Toxicology (SOT) 54<sup>th</sup> Annual Meeting, San Diego, CA, March 22-26.

Zu, K; Tao, G; Kerper, LE; Rhomberg, LR; Goodman, JE. 2015. "Evaluation of US EPA's Noncancer Risk Assessment of Libby Amphibole Asbestos." *Toxicologist* 144(1):164. Abstract 768. Presented at the Society of Toxicology (SOT) 54<sup>th</sup> Annual Meeting, San Diego, CA, March 22-26.

**Julie E. Goodman, Ph.D., DABT, FACE, ATS**

Sax, SN; Pizzurro, DM; Zu, K; Lynch, HN; Prueitt, RL; Goodman, JE. 2015. "Ozone Exposure and Systemic Biomarkers: Evaluation of Evidence of Adverse Cardiovascular Health Impacts." *Toxicologist* 144(1):166-167. Abstract 778. Presented at the Society of Toxicology (SOT) 54<sup>th</sup> Annual Meeting, San Diego, CA, March 22-26.

Prueitt, RL; Lynch, HN; Tabony, JA; Beck, NB; Goodman, JE; Rhomberg, LR 2015. "Evaluation of Study Quality Criteria Frameworks." *Toxicologist* 144(1):400. Abstract 1865. Presented at the Society of Toxicology (SOT) 54<sup>th</sup> Annual Meeting, San Diego, CA, March 22-26.

Kerper, LE; Lynch, HN; Zu, K; Tao, G; Utell, M; Goodman, JE. 2015. "A Systematic Review of Asbestos-induced Pleural Plaques and Lung Function." *Toxicologist* 144(1):401. Abstract 1869. Presented at the Society of Toxicology (SOT) 54<sup>th</sup> Annual Meeting, San Diego, CA, March 22-26.

Seeley, MR; Thakali, S; Goodman, JE. 2015. "Birth Defect Rates in High and Low Atrazine-use States." *Toxicologist* 144(1):462. Abstract 2151. Presented at the Society of Toxicology (SOT) 54<sup>th</sup> Annual Meeting, San Diego, CA, March 22-26.

Loftus, CT; Goodman, JE; Zu, K. 2015. "2,4-dichlorophenoxyacetic Acid and Non-Hodgkin's Lymphoma, Gastric Cancer, and Prostate Cancer: Meta-analyses of the Published Literature." *Toxicologist* 144(1 (Supplement)):192. Abstract 2831. Presented at the Society of Toxicology (SOT) 54<sup>th</sup> Annual Meeting, San Diego, CA, March 22-26.

Goodman, JE; Lynch, HN; Prueitt, RL; Beck, NB; Tabony, JA; Rhomberg, LR. 2014. "Evaluation of Study Quality Criteria Frameworks." Presented at the Society for Risk Analysis Annual Meeting, Denver, Colorado, December 7-10.

Sax, SN; Pizzurro, DM; Zu, K; Lynch, HN; Prueitt, RL; Goodman, JE. 2014. "Weight-of-evidence Evaluation of Short-term Ozone Exposure and Cardiovascular Biomarkers." Presented at the Society for Risk Analysis Annual Meeting, Denver, Colorado, December 7-10.

Lange, SS; Rhomberg, LR; Dourson, ML; Tao G, Goodman JE, Honeycutt M. 2014. "How Dose Response Curves Derived from Clinical Ozone Exposures can Inform Public Policy." Presented at the Society for Risk Analysis Annual Meeting, Denver, Colorado, December 7-10.

Kerper, LE; Lynch, HN; Mohr, LC; Goodman, JE. 2014. "Do Asbestos-induced Pleural Plaques Cause Lung Function Deficits?" *Toxicologist* 138(1):475. Abstract 1811. Presented at the Society of Toxicology (SOT) 53<sup>rd</sup> Annual Meeting, Phoenix, AZ, March 23-27.

Prueitt, RL; Sax, SN; Lynch, HN; Lemay, JC; King, JM; Goodman, JE. 2014. "Weight-of-evidence Evaluation of Short-term Ozone Exposure and Cardiovascular Effects." *Toxicologist* 138(1):475. Abstract 1810. Presented at the Society of Toxicology (SOT) 53<sup>rd</sup> Annual Meeting, Phoenix, AZ, March 23-27.

Lemay, JC; Prueitt, RL; Hixon, ML; Goodman, JE. 2013. "Distinguishing between Risks and Hazards: A Case Study of Bisphenol A." Presented at the Society for Risk Analysis Annual Meeting, Baltimore, MD, December 8-10. 13p.

Sax, SN; Prueitt, RL; Goodman, JE. 2013. "Weight-of-evidence Evaluation of Short-term Ozone Exposure and Cardiovascular Effects." Presented at the Society for Risk Analysis Annual Meeting, Baltimore, MD, December 8-11.

**Julie E. Goodman, Ph.D., DABT, FACE, ATS**

Goodman, JE, Prueitt RL, Rhomberg, LR. 2013. "Hypothesis-based Weight-of-evidence Evaluation of the Human Carcinogenicity of Toluene Diisocyanate." Presented at the Conference on Isocyanates and Health: Past, Present and Future, April 3-4.

Seeley, MR; Goodman, JE. 2013. "Is There a Subset of Susceptible Individuals in Controlled Air Pollution Studies?" *Toxicologist* 132(1):414. Abstract No. 1945. Presented at the Society of Toxicology (SOT) 52<sup>nd</sup> Annual Meeting, San Antonio, TX, March 10-14.

Prueitt, RL; Goodman, JE; Rhomberg, LR. 2013. "Hypothesis-based Weight-of-evidence Evaluation of the Human Carcinogenicity of Toluene Diisocyanate." *Toxicologist* 132(1):415. Abstract No. 1951. Presented at the Society of Toxicology (SOT) 52<sup>nd</sup> Annual Meeting, San Antonio, TX, March 10-14.

Rhomberg, LR; Goodman, JE; Bailey, EA; Prueitt, RL. 2012. "Weight-of-evidence Frameworks, Systems, and Tools: A Survey of Existing Approaches and Notes on Best Practices." Presented at the "Putting it All Together: Recent Developments in Risk Assessment Approaches" Symposium, Society for Risk Analysis Annual Meeting, San Francisco, CA, December 11. 24p.

Sax, SN; Lau, JH; Goodman, JE. 2012. "Evaluation of the BenMAP Model for Estimating Mortality Impacts of Lower Ozone Concentrations." Presented at the 22<sup>nd</sup> Annual Conference of the International Society of Exposure Science, Seattle, WA, October 28-November 1.

Bailey, LA; Goodman, JE; Beck, BD. 2012. "Revised Reference Concentration for Manganese Oxide Based on Recent Epidemiology and Pharmacokinetic Studies." *Toxicologist* 126(1):213. Abstract No. 995. Presented at the Society of Toxicology (SOT) 51<sup>st</sup> Annual Meeting, San Francisco, CA, March 11-15.

Peterson, MK; Goodman, JE. 2012. "Infant Risk and Exposure Assessment of Bisphenol A in Polycarbonate and 'BPA-free' Plastic Bottles." *Toxicologist* 126(1):319. Abstract No. 1478. Presented at the Society of Toxicology (SOT) 51<sup>st</sup> Annual Meeting, San Francisco, CA, March 11-15.

Prueitt, RL; Goodman, JE; Bailey, LA; Rhomberg, LR. 2012. "Hypothesis-based Weight-of-evidence Evaluation of the Neurodevelopmental Effects of Chlorpyrifos." *Toxicologist* 126(1):309. Abstract No. 1430. Presented at the Society of Toxicology (SOT) 51<sup>st</sup> Annual Meeting, San Francisco, CA, March 11-15.

Seeley, MR; Goodman, JE. 2012. "Is Sulfur Dioxide a Reproductive and Developmental Toxicant?" *Toxicologist* 126(1):24. Abstract No. 119. Presented at the Society of Toxicology (SOT) 51<sup>st</sup> Annual Meeting, San Francisco, CA, March 11-15.

Bailey, LA; Goodman, JE; Rhomberg, LR. 2011. "Hypothesis-based Weight-of-evidence Evaluation of Naphthalene: Carcinogenic Hazard Assessment and Mode of Action." Presented at SETAC North America 32<sup>nd</sup> Annual Meeting, Boston, MA, November 14. 1p.

Prueitt, RL; Goodman, JE. 2011. "Evaluation of Adverse Effects on Human Lung Function Caused by Ozone." *Toxicologist* 120(Suppl. 2):491. Abstract No. 2286. Presented at the Society of Toxicology (SOT) 50<sup>th</sup> Annual Meeting, Washington, DC, March 6-11.

Peterson, MK; Bailey, LA; Dodge, DG; Goodman, JE; Valberg, PA. 2011. "A Weight-of-evidence Evaluation of Asbestos Exposure and Mesothelioma Risk among Electricians." *Toxicologist* 120(Suppl. 2):414. Abstract No. 1935. Presented at the Society of Toxicology (SOT) 50<sup>th</sup> Annual Meeting, Washington, DC, March 6-11.

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Goodman, JE; Mayfield, DB; Bailey, LA; Rhomberg, LR. 2011. "Weight-of-evidence Evaluation of Formaldehyde Exposure and Leukemia Risk." *Toxicologist* 120(Suppl. 2):416. Abstract No. 1944. Presented at the Society of Toxicology (SOT) 50<sup>th</sup> Annual Meeting, Washington, DC, March 6-11.

Mattuck, RL; Seeley, MR; Reid, KR; Goodman, JE. 2011. "Human Health Risks from Exposure to 1,4-butanediol in Craft Kit Beads." *Toxicologist* 190(Suppl. 2):332. Abstract No. 1545. Presented at the Society of Toxicology (SOT) 50<sup>th</sup> Annual Meeting, Washington, DC, March 6-11.

Dodge, DG; Pollock, MC; Sax, SN; Petito Boyce, C; Goodman, JE. 2011. "Risk Characterization of the Brominated Flame Retardant Decabromodiphenyl Ethane in Indoor Dust." *Toxicologist* 120(Suppl. 2):271. Abstract No. 1268. Presented at the Society of Toxicology (SOT) 50<sup>th</sup> Annual Meeting, Washington, DC, March 6-11.

Peterson, MK; Bailey, LA; Dodge, DG; Goodman, JE; Valberg, PA. 2010. "Risk Assessment of Mesothelioma Among Electricians." Presented at the Society for Risk Analysis Annual Meeting, Salt Lake City, UT, December 5-8.

Haber, LT; Prueitt, RL; Goodman, JE; Thakali, S; Patterson, J. 2010. "Report of a Workshop: An Evaluation of Hypotheses for Determining the Carcinogenic Potential of Nickel-containing Substances." Presented at the Society for Risk Analysis Annual Meeting, Salt Lake City, Utah, December 5-8. 1p.

Goodman, JE; Kerper, LE; Petito Boyce, C; Prueitt, RL; Rhomberg, LR. 2010. "Weight of Evidence Exposures Analysis of Exposures to Dioxins and Dioxin-like Compounds and Associations with Thyroid Hormone Levels during Early Development." Presented at the Society of Toxicology (SOT) 49<sup>th</sup> Annual Meeting, Salt Lake City, UT. 1p.

Prueitt, RL; Goodman, JE; Thakali, S. 2010. "An Evaluation of Hypotheses for Determining the Carcinogenic Potential of Nickel-containing Substances." Presented at the Society of Toxicology (SOT) 49<sup>th</sup> Annual Meeting, Salt Lake City, UT. 1p.

Dodge, DG; Goodman, JE; Beck, BD. 2010. "Weight-of-evidence Analysis of Hydroquinone and Leukemia." *Toxicologist* 114(1):111-112. Abstract No. 514. Presented at the Society of Toxicology (SOT) 49<sup>th</sup> Annual Meeting, Salt Lake City, UT, March 7-11.

Thakali, S; Chandalia, JK; Seeley, M; Goodman, JE. 2010. "Meta-analysis of Nitrogen Dioxide Effects on Airway Hyper-responsiveness in Asthmatics: Effects of the Types of Airway Challenge, Exposure Methods, and Activities During Exposure." *Toxicologist* 114(1):401. Abstract No. 1886. Presented at the Society of Toxicology (SOT) 49<sup>th</sup> Annual Meeting, Salt Lake City, UT, March 7-11.

Goodman, JE; Chandalia, JK; Thakali, S; Seeley, M. 2009. "Meta-analysis of Controlled Nitrogen Dioxide Exposure Studies Assessing Airway Hyper-responsiveness in Asthmatics." Presented at the Society for Risk Analysis Annual Meeting, Baltimore, MD, December 6-9.

Goodman, JE; Rhomberg, LR; Prueitt, RL. 2009. "A Weight-of-evidence Analysis of the Human Carcinogenicity of Styrene." Presented at the Annual Meeting of the American College of Epidemiology, Silver Spring, MD, September 13-15.

Prueitt, RL; Goodman, JE; Dodge, DG; Thakali, S. 2009. "A Weight-of-evidence Evaluation of the Carcinogenicity of Soluble Nickel." *Toxicologist* 108(1):328. Abstract No. 1582. Presented at the Society of Toxicology (SOT) 48<sup>th</sup> Annual Meeting, Baltimore, MD, March 15-19.

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Goodman, JE; Rhomberg, LR. 2009. "A Weight-of-evidence Approach to Evaluating Epidemiological Data on Styrene Cancer Hazards." *Toxicologist* 108(1):248. Abstract No. 1190. Presented at the Society of Toxicology (SOT) 48<sup>th</sup> Annual Meeting, Baltimore, MD, March 15-19.

Beyer, LA; Beck, BD; Goodman, JE. 2009. "Background Rates of Lymphomas/Leukemias and Leydig Cell Tumors in Sprague Dawley Rats." *Toxicologist* 108(1):421. Abstract No. 2028. Presented at the Society of Toxicology (SOT) 48<sup>th</sup> Annual Meeting, Baltimore, MD, March 15-19.

Goodman, JE; Bailey, LA; Beck, BD. 2008. "Recent Occupational Studies of Manganese and their Bearing on the Reference Concentration (RfC)." Presented at the Annual Meeting of the American College of Epidemiology, Tucson, AZ, September 14-16.

Aylward, LL; Goodman, JE; Charnley, G; Rhomberg, LR. 2008. "A Margin of Exposure Approach to Assessment of Non-cancer Risks of Dioxins Based on Human Exposure and Response Data." Presented at the 28<sup>th</sup> International Symposium on Halogenated Persistent Organic Pollutants, Birmingham, England, August 17-22.

Goodman, JE; Bailey, LA; Beck, BD. 2008. "Recent Studies of the Health Effects of Manganese and the Implications for the Reference Concentration (RfC)." *Toxicologist* 102. Abstract No. 1787. Presented at the Society of Toxicology (SOT) 47<sup>th</sup> Annual Meeting, Seattle, WA, March 16-20.

Dodge, DG; Haber, LT; Kopras, E; Goodman, JE; Pagan, I; Gift, JS; Rhomberg, LR. 2008 "Case Studies for the Development of a Pathophysiological Progression Model." *Toxicologist* 102(1):245. Abstract No. 1189. Presented at the Society of Toxicology (SOT) 47<sup>th</sup> Annual Meeting, Seattle, WA, March 16-20.

Beyer, LA; Slayton, TM; Goodman, JE; Greenberg, GI; Hudson, TC; Sax, SN; Beck, BD. 2008 "Evaluation of Key Information Informing the Basis of EPA's New Recommended Ozone Standard." *Toxicologist* 102. Abstract No. 1462. Presented at the Society of Toxicology (SOT) 47<sup>th</sup> Annual Meeting, Seattle, WA, March 16-20.

Pagan, I; Haber, LT; Rhomberg, LR; Goodman, JE; Dodge, DG; Foureman, GL. 2007. "Development of a Pathophysiological Progression Model for Selected Endpoints." Presented at the Society for Risk Analysis Annual Meeting, San Antonio, TX, December 9-12.

Haber, LT; Rhomberg, LR; Goodman, JE; Dodge, DG; Zhao, QJ; Pagan, I; Foureman, GL. 2007. "Considerations Regarding the Structure and Application of a Pathophysiological Progression Model." Presented at the Society for Risk Analysis Annual Meeting, San Antonio, TX, December 9-12.

Dodge, DG; Zhao, QJ; Haber, LT; Goodman, JE; Pagan, I; Foureman, GL; Rhomberg, LR. 2007. "Case Studies for the Development of a Pathophysiological Progression Model: Fatty Liver." Presented at the Society for Risk Analysis Annual Meeting, San Antonio, TX, December 9-12.

Mechanic, LE; Luke, BT; Goodman, JE; Chanock, SJ; Harris, CC. 2007. "Polymorphism Interaction Analysis (PIA): A Method for Investigating Complex Gene-gene Interactions." Presented at Approaches to Complex Pathways in Molecular Epidemiology, Santa Ana Pueblo, NM, May 30-June 2.

Goodman, JE; Gaylor, D; Beyer, LA; Rhomberg, LR; Beck, BD. 2007. "MTBE is Not Associated with a Statistically Significant Increase in Leydig Cell Tumors in Sprague-Dawley Rats." *Toxicologist* 96(1):339. Abstract No. 1637. Presented at the Society of Toxicology (SOT) 46<sup>th</sup> Annual Meeting, Charlotte, NC, March 25-29.

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Rhomberg, LR; Goodman, JE; McConnell, EE; Sipes, IG; Witorsch, RJ; Slayton, TM; Yu, CJ; Lewis, AS. 2007. "An Updated Weight of the Evidence Evaluation of Reproductive and Developmental Effects of Low Doses of Bisphenol A." *Toxicologist* 96(1):427. Abstract No. 2067. Presented at the Society of Toxicology (SOT) 46<sup>th</sup> Annual Meeting, Charlotte, NC, March 25-29.

Beyer, LA; Goodman, JE; Seeley, MR; Slayton, TM; Beck, BD. 2007. "Carcinogenicity Evaluation of Methyl Tert-butyl Ether (MTBE)." *Toxicologist* 96(1):325. Abstract No. 1569. Presented at the Society of Toxicology (SOT) 46<sup>th</sup> Annual Meeting, Charlotte, NC, March 25-29.

Zanetti, KA; Kahn, MA; Bowman, ED; Goodman, JE; Chanock, S; Harris, CC. 2007. "Compromised Complement System Increases Colon Cancer Susceptibility in African-Americans." *Proc. Am. Assoc. Cancer Res.* 48.

Schoen, A; Eldan, M; Goodman, JE; Beck, BD. 2006. "DMAV-induced Bladder Tumors: Unique Rat Susceptibility." *Toxicologist* 90(1):448. Abstract No. 2186. Presented at the Society of Toxicology (SOT) 45<sup>th</sup> Annual Meeting, San Diego, CA, March 5-9.

Goodman, JE; Harris, CC. 2005. "GST-T1, p53, and CASPASE-8 Polymorphisms and Colon Cancer Risk." Presented at the Society of Toxicology (SOT) 44<sup>th</sup> Annual Meeting, New Orleans, LA, March 6-10.

Goodman, JE; Bowman, E; Chanock, S; Harris, CC. 2004. "Arachidonate Lipoxygenase (ALOX) and Cyclooxygenase (COX) Polymorphisms and Colon Cancer Risk." *Proc. Am. Assoc. Cancer Res.* 44.

Goodman, JE; Bowman, E; Chanock, S; Harris, CC. 2004. "ALOX & COX Polymorphisms & Colon Cancer Risk: Association with ALOX-5 G-1753A & G-1700A." Center for Cancer Research Fourth Annual Fellows and Young Investigators Retreat, Williamsburg, VA, March 9-11.

Sullivan, AE; Goodman, JE; Silber, PM; Yager, JD. 2004. "Correlation Between Catechol-O-methyltransferase Genotype and Phenotype." *Toxicologist* 88.

Sullivan, AE; Goodman, JE; Yager, JD. 2003. "Catechol-O-methyltransferase (COMT) and Catechol Estrogens in Breast Cancer." Presented at the 226<sup>th</sup> National American Chemical Society Division of Toxicology Meeting, New York, NY, September 7-11.

Goodman, JE; Sullivan, AE; He, P; Silber, PM; Yager, JD. 2003. "Correlation Between Catechol-O-methyltransferase Genotype and Phenotype." AACR Molecular and Genetic Epidemiology of Cancer Conference Proceedings, Waikoloa, HI, January 18-23.

Goodman, JE; He, P; Yager, JD. 2002. "COMT Polymorphism and Catechol Estrogen Methylation in Breast Epithelial Cell Lines." *Proc. Am. Assoc. Cancer Res.* 45.

Goodman, JE; He, P; Yager, JD. 2001. "Kinetics of High and Low Activity Human Catechol-O-methyltransferase Activity for Catechol Estrogen Methylation." *Proc. Am. Assoc. Cancer Res.* 42.

Lavigne, JA; Goodman, JE; Fonong, T; Odwin-DeCosta, S; He, P; Yager, JD. 2001. "The Effects of Catechol-O-methyltransferase Inhibition on Catechol Estrogen Levels and Oxidative DNA Damage in MCF-7 Cells." *Proc. Am. Assoc. Cancer Res.* 42.

Goodman, JE; Lavigne, JA; Hengstler, JG; Helzlsouer, KJ; Yager, JD. 2000. "Catechol-O-methyltransferase Polymorphism Not Associated with Ovarian Cancer." *Proc. Am. Assoc. Cancer Res.* 41.

**Julie E. Goodman, Ph.D., DABT, FACE, ATS**

Chen, JQ; Delannoy, M; Goodman, JE; Lavigne, JA; Odwin, SE; He, P; Yager, JD. 2000. "Enhanced Transcript Levels of Mitochondrial Genes, Respiratory Chain Activity, Bcl-2 Levels and Glutathione Distribution by Ethinyl Estradiol in Female Rat Hepatocytes." *Proc. Am. Assoc. Cancer Res.* 41.

**Invited Lectures, Testimony, and Other Presentations**

"A Case for Good Epidemiology Practice Guidelines for Regulatory Risk Assessment." Presented at the Environmental Epidemiology Committee Webinar, September 23, 2021.

"Food Safety." Presented at Gradient's Trends 75 Webinar, June 19, 2019.

"Evaluating Adverse Drug Effects in Pharmacoepidemiology Studies." Presented at Gradient's Trends 71 Webinar, February 28, 2018.

"Study Quality and Evidence Integration in the IRIS Process." Presented at the National Academies of Science, Engineering, and Medicine Review of Advances Made to the IRIS Process Workshop, Washington, DC, February 1, 2018.

"Challenges for the Agrochemical Industry." Presented at the International Society of Exposure Science (ISES) Annual Conference, Research Triangle Park, NC, October 17, 2017.

"National Ambient Air Quality Standards." Presented at the Institute for Humane Studies and the Mercatus Center Risk Analysis Seminar, Portland, OR, June 27, 2016.

"Why Epidemiologists Need Toxicologists (and Vice Versa)." Presented at the CropLife America & RISE 2016 Spring Conference, Arlington, VA, April 14, 2016.

"Systematic Review." Presented at the Asphalt Institute Spring Meeting, Nashville, TN, April 13, 2016.

"An Introduction to Meta-analysis." Presented at the Johns Hopkins Bloomberg School of Public Health, Baltimore, MD, April 5, 2016.

"Evaluation of Scoring Approaches and Consideration of an Independent Approach." Presented at the 40<sup>th</sup> Annual Winter Meeting of Toxicology Forum, Washington, DC, February 10, 2016.

"Extrapolation of Controlled Human Study Results to the US Population." Presented at the Society for Risk Analysis Annual Meeting, December 6-9, 2015.

"How a Sensitivity Analysis of Raw Data Would Strengthen EPA's Chlorpyrifos Risk Assessment." Presented at the Society for Risk Analysis Annual Meeting, December 6-9, 2015.

"The Future of Toxicology." Presented at the SPI Food Packaging Summit, November 10, 2015.

"Evaluation of Evidence EPA Cites in Support of the Ozone National Ambient Air Quality Standards for Ozone Proposed Rule." Presented at the 2015 Env-vision Conference, May 12-14, 2015.

"The Scientific Evidence Does Not Support the Administrator's Proposed Conclusion that the Primary Ozone NAAQS Should Be Between 0.065 and 0.070 ppm." Testimony at the US Environmental Protection Agency (EPA) Public Hearing on the Proposed Updates to the National Ambient Air Quality Standards (NAAQS) for Ground-level Ozone, Washington, DC, January 29, 2015.

**Julie E. Goodman, Ph.D., DABT, FACE, ATS**

"Risk-of-bias Analysis: Case Study of Pleural Plaques and Lung Function." Presented at the Society for Risk Analysis Annual Meeting, December 7-10, 2014.

"Is a Stricter Ozone NAAQS Supported by the Science?" Presented at the Association of Battery Recyclers 2014 Fall Meeting, October 16, 2014.

"NexGen: Are We There Yet?" Presented at the 2014 Health Effects Institute Annual Conference, May 6, 2014.

"Hypotheses and Weight-of-evidence Frameworks." Presented at the 53<sup>rd</sup> Annual Society of Toxicology Meeting, March 23, 2014.

"When an Association Indicates Causation." Presented at the 53<sup>rd</sup> Annual Society of Toxicology Meeting, March 23, 2014.

"Rethinking Meta-analysis: Applications for Air Pollution Data and Beyond." Presented at the Society for Risk Analysis Annual Meeting, December 8-11, 2013.

"Incorporation of Weight-of-evidence Best Practices in the National Ambient Air Quality Standards Review Process." Presented at the Society for Risk Analysis Annual Meeting, December 8-11, 2013.

"Bradford Hill Viewpoints and Hypothesis-based Weight of Evidence." Presented at the Society for Risk Analysis Annual Meeting, December 8-11, 2013.

"Rethinking Meta-analysis: Applications for Air Pollution Data and Beyond." Presented at the Harvard Systematic Review Symposium, October 3-4, 2013.

"A Hypothesis-based Weight-of-evidence Approach to Evaluate the Human Carcinogenicity of Isocyanates." Presented at the Isocyanates & Health Conference, April 3, 2013.

"Using Epidemiology to Analyze Neurodevelopmental Toxicity Across Species." Presented at the 52<sup>nd</sup> Annual Society of Toxicology Meeting, March 14, 2013.

"Designing Case-control Studies." Presented at the New York Medical College School of Health Science & Practice, February 21, 2013.

"Designing Cohort Studies." Presented at the New York Medical College School of Health Science & Practice, February 11, 2013.

"Systemic Reviews and Meta-analysis." Presented in the Use of Expert Elicitation to Inform Decisionmaking Workshop, Society for Risk Analysis 2012 Annual Meeting, San Francisco, CA, December 2012.

"Survival Analysis & Meta-analysis." Presented at the New York Medical College School of Health Sciences & Practice, November 8, 2012.

"Overview of the Controversy Surrounding Bisphenol A Toxicity." Presented at the Biological Relevance and Health Concerns of Genotoxicity Conference, Newark, DE, October 24, 2012.

"Biological & Statistical Interaction." Presented at the New York Medical College School of Health Sciences & Practice, October 11, 2012.

**Julie E. Goodman, Ph.D., DABT, FACE, ATS**

Testimony regarding "EPA's Assessment of Health Benefits Associated with PM<sub>2.5</sub> Reductions for the Final Mercury and Air Toxics Standards." Presented to the Subcommittee on Energy and Power, United States Congressional Committee on Energy and Commerce American Energy Initiative Hearing, Washington, DC, February 8, 2012.

"Synthesizing Evidence: An Introduction to Systematic Reviews, Meta-analysis, and Expert Elicitation." Presented at the Society for Risk Analysis 2011 Annual Meeting, Charleston, SC, December 2011.

"Why Meta-analyses and Systematic Reviews Come to Different Conclusions About Formaldehyde and Leukemia." Presented at the Society for Risk Analysis 2011 Annual Meeting, Charleston, SC, December 2011.

"The Weight of Evidence Regarding Bisphenol A and Human Health." Presented at the Society for Risk Analysis New England Chapter Meeting, Harvard School of Public Health, Boston, MA, December 2011.

Testimony regarding Air Quality and Children's Health. Presented to the Subcommittee on Clean Air and Nuclear Safety and the Subcommittee on Children's Health and Environmental Responsibility, United States Senate Committee on Environment and Public Works Hearing, Washington, DC, June 8, 2011.

"On Babies, Bottles, and Bisphenol A." Presented at the Defense Research Institute Toxic Torts and Environmental Law Seminar, New Orleans, LA, February 2011.

"Bisphenol A and Human Health: What Does the Science Show?" Presented at the Policy of BPA Event, American Enterprise Institute, Washington, DC, June 2010.

"Human Health Risk Assessment." Presented at the Massachusetts Maritime Academy, Buzzards Bay, MA, May 2010.

"The Science Behind the Reconsideration of the Ozone NAAQS." Presented as part of the webinar, How Will EPA's New Ozone Standards Affect Your Community? April 2010.

"Weight-of-evidence Analysis of Reproductive and Developmental Health Effects of Bisphenol A." Presented at the Nypro Bisphenol A Information Event, Clinton, MA, March 2010.

"Everyday Exposures to Bisphenol A Do Not Cause Adverse Health Effects in Humans." Presented at the Harvard Extension School, Environmental Management Program, Cambridge, MA, March 2010.

"New Developments in Exploratory Research on 'Estrogenicity' – Progress Toward Validation of New Endpoints and Testing Methods." Presented at the Society of the Plastics Industry's Food, Drug, and Cosmetic Packaging Materials Committee Winter Conference, Atlanta, GA, December 2009.

"Avoiding Potential Long-term Liability through Risk Assessment for Material Selection." Presented at the 21<sup>st</sup> Annual Product Liability Conference, University of Wisconsin-Madison, Madison, WI, September 2009.

"Epidemiology and Risk Assessment." Presented at the Annual Meeting of the American College of Epidemiology, Silver Spring, MD, September 2009.

"Risk Assessment Techniques for Materials Selection." Presented as part of the Strategies for Substance Replacement in Products Webinar, May 2009.

**Julie E. Goodman, Ph.D., DABT, FACE, ATS**

"Investigation of Potential Cancer Clusters in Northampton, MA." Presented at the John F. Kennedy School, Northampton, MA, September 2008.

"Did Chemicals in Your Product Cause John Doe's Disease? The Toxicologist Speaks." Presented at the 20<sup>th</sup> Annual Product Liability Conference, University of Wisconsin-Madison, Madison, WI, September 2008.

"Investigation of Potential Disease Clusters in Northampton, MA: Progress Update." Presented at the Robert K. Finn Ryan Road School, Northampton, MA, October 2007.

"Investigation of Potential Disease Clusters in Northampton, MA." Presented at the Robert K. Finn Ryan Road School, Northampton, MA, May 2007.

"Single Nucleotide Polymorphisms (SNPs), Inflammation and Colon Cancer." Presented at the Cancer Prevention Fellowship Program Seminar Series, NCI, Rockville, MD, February 2004.

"The Epidemiology of Inflammation and Colon Cancer." Presented at the Laboratory of Human Carcinogenesis Meeting, NCI, Bethesda, MD, December 2003.

"Macrophage Migration Inhibitory Factor (MIF) in Inflammatory Bowel Diseases." Presented at the Laboratory of Human Carcinogenesis International Workshop, Bethesda, MD, September 2003.

"Chronic Inflammation and Colon Cancer Risk." Presented at the Cancer Prevention Fellowship Program Seminar Series, NCI, Rockville, MD, June 2003.

## Appendix B

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**Testimony Experience (2019-2023) of Julie E. Goodman,  
Ph.D., DABT, FACE, ATS**

**Expert Testimony**  
**Julie E. Goodman, Ph.D., DABT, FACE, ATS**  
**through August 2023**

Expert #	For:	Plaintiff (P)	Defendant (D)	Case #	Court	District	Date	Legal Proceeding	Protected?
1	D	Theresa Anderson	Ford Motor Company	18-CV-251	Circuit Court	State of Wisconsin, County of Lacrosse	10/23/19	Deposition	No
2	D	Amber, <i>et al.</i>	Allied Waste Transportation, Inc., <i>et al.</i>	09 L 15741	Circuit Court	State of Illinois, County of Cook	10/29/19	Deposition	No
3	D	Véronique Lalande and Louis Duchesne	Quebec Stevedoring Company Limited and Quebec Port Authority	200-06-000169-139	Superior Court	Province of Quebec, District of Quebec	12/10/19	Trial	No
4	D	Estate of William Hale	City of Portland and/or Maine Municipal Association	06033944	Worker's Compensation Board		7/7/20	Deposition	No
5	D	Marc Czapla and Jill Czapla	Republic Services, Inc., <i>et al.</i>	18SL-CC00803-01	Circuit Court	State of Missouri, County of St. Louis	10/30/20	Deposition	No
6	D	Steven Sarkis and Judy Sarkis	Advance Stores Company Incorporated, <i>et al.</i>	Civil Action No. 19-3312	Superior Court	State of Massachusetts, County of Middlesex	11/12/20	Deposition	No
7	D	Amber, <i>et al.</i>	Allied Waste Transportation, Inc., <i>et al.</i>	09 L 15741	Circuit Court	State of Illinois, County of Cook	6/16/21	Deposition	No
8	D	Estate of Wayne D. Vetre	Town of Wells/Maine Municipal Association	218W2797	Worker's Compensation Board		12/14/21	Deposition	No
9	D	John C. Riegler and Kathi A. Riegler	Ford Motor Company	2:20-cv-00752-RJS-CMR	District Court	District of Utah	4/6/2022	Deposition	No
10	D	Pamela S. Rud and David Rud	Ford Motor Company	21-L-286	Circuit Court	State of Illinois, County of Madison	5/24/2022	Deposition	No
11	D	Estate of Eugene G. Hohlfeld, Sr.	Ford Motor Company, <i>et al.</i>	18-CV-251	Circuit Court	State of Wisconsin, County of Lacrosse	9/14/2022	Trial	No
12	D	Estate of Vincent DiFillipo, Sr.	City of Portland and Maine Municipal Association		Worker's Compensation Board		9/29/2022	Deposition	No
13	D	District of Columbia	Beech-Nut Nutrition Company	2021 CA 001292B	Superior Court	District of Columbia	3/2/2023	Deposition	No
14	D	Citizens for the Environment	Elcon Recycling Center (2003) Ltd., <i>et al.</i>	CA 36568-07-19	District Court	Haifa, Israel	5/15/2023; 5/17/2023	Trial	No
15	D	Vicki Lee and the Estate of Steven A. Lee	Ford Motor Company	2019CV000344	Circuit Court	State of Wisconsin, County of Grant	6/1/2023	Deposition	No
16	D	Robert Collins	Allied Fluid Products Corp., <i>et al.</i>	22CV021614	Superior Court	State of California, County of Alameda	6/5/2023	Deposition	No
17	D	Bryan Dick-Ipsen	Tri-Supply Co., <i>et al.</i>	2018-L-011367	Circuit Court	State of Illinois, Cook County	8/10/2023	Deposition	No

## Appendix C

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### List of Documents Considered

**Plaintiff Documents:**

- Candice Cherrybone Deposition Transcript with Subject Matter Index.pdf
- 2023.08.16 Candice Cherrybone First Written Discovery Responses.pdf
- 2023.09.08 New Indy HIPAA- Cherrybone, Candice-Executed.pdf
- Melda Gain Deposition Transcript with Subject Matter Index.pdf
- 2023.09.08 Plaintiff Melda Gain's Supplemental Answers to Defendant's First Written Discovery (redlined).pdf
- 2023.09.07 New Indy HIPAA - M Gain - Executed.pdf
- 2023.09.08 Melda Gain Declaration Page.docx.pdf
- 2023.09.08 Melda Gains Answer to 3rd Discovery.pdf
- 2023.09.08 Melda Gain's Supplemental Answers to Defendant's First Written Discovery.pdf
- 2023.08.30 Melda Gains Answer to 3rd Discovery.pdf
- 2023.03.17 Melda Gain Answers to Defs' Second Discovery.pdf
- Melda Gain Answers to Defs' Second Discovery.pdf
- 2022.11.02 Melda Gain's Responses to Defendants' First Written Discovery.pdf
- 2023.02.17 Plaintiff Melda Gain's Supplemental Answers to Defendant's First Written Discovery.pdf
- Orrin Gain Deposition Transcript with Subject Matter Index.pdf
- 07.17.23 Orrin Gain Declaration Page - executed.pdf
- 2023.07.17 Orrin Gain First Written Discovery Responses.pdf
- 2023.08.30 Orrin Gain's Answers to 2nd Discovery.pdf
- 2023.09.07 Orrin Gain Supplemental Written Discovery Responses.pdf
- 2023.09.08 Orrin Gain Declaration Page.docx (1).pdf
- 2023.09.08 Orrin Gain Supplemental Written Discovery Responses (redlined).pdf
- 2023.09.08 Orrin Gain Supplemental Written Discovery Responses.pdf
- 2023.09.08 Orrin Gain's Answers to 2nd Discovery.pdf
- Marty Kennedy Deposition Transcript with Subject Matter Index.pdf
- M. Kennedy Medical Questionnaire.pdf
- 07.17.23 Marty Kennedy Declaration Page - executed.pdf
- 2023.07.17 Marty Kennedy First Written Discovery Responses.pdf
- 2023.08.24 Marty Kennedy Supplemental Discovery Responses.pdf
- 2023.09.07 New Indy HIPAA-Kennedy, Marty-Executed.pdf
- T. Kennedy Medical Questionnaire.pdf
- Terri Kennedy Deposition Transcript with Subject Matter Index.pdf
- 23.09.08 Plaintiff Terri Kennedy's Supplemental Written Discovery.pdf

- 2023.09.07 New Indy HIPAA-Kennedy, Terri-Executed.pdf
- 2023.09.08 Kennedy Facebook Screen Shots\_9.8.23\_.pdf.pdf
- 2023.09.08 Plaintiff Terri Kennedy's Supplemental Written Discovery.pdf
- 2023.09.08 Terri Kennedy Declaration Page.docx 2.pdf
- 2023.03.13 Terri Kennedy Answers to Defs' Second Discovery.pdf
- 2023-09-08 Plaintiff Terri Kennedy's Supplemental Written Discovery (redlined).pdf
- 2023.03.17 Terri Kennedy Answers to Defs' Second Discovery.pdf
- Terri Kennedy Answers to Defs' Second Discovery.pdf
- 2022.11.02 Terri Kennedy's Responses to Defendants' First Written Discovery.pdf
- 2023.02.17 Plaintiff Terri Kennedy's Supplemental Answers to Defendant's First Written Discovery.pdf
- Kennedy\_0000005.pdf
- E. Lizano Deposition Transcript with Subject Matter Index.pdf
- E. Lizano Medical Questionnaire.pdf
- 2023.09.08 Enrique Lizano's Sup. Answer to Def's Second Discovery.pdf
- 2023.09.05 New Indy HIPAA - E Lizano - Executed.pdf
- 2023.09.08 Enrique Lizano Declaration Page.pdf
- 2023.09.08 Enrique Lizano's Sup. Answer to Def's Second Discovery (redlined).pdf
- 2023.01.20 E. Lizano Resp to 2nd Set of Discovery.pdf
- 2023.05.12 Enrique Lizano's Sup. Answer to Def's Second Discovery.pdf
- 2022.11.02 Enrique Lizano's Answers to Defendants' First Written Discovery.pdf
- 2023.02.17 Plaintiff Enrique Lizano's Supplemental Answers to Defendant's First Written Discovery.pdf
- Lizano 0000001 (1).xlsx
- Lizano 0000005.pdf
- Lizano 0000007.pdf
- Lizano 0000116.pdf
- Lizano 0000226.xlsx
- Lizano 0000327.pdf
- Lizano 0000328.pdf
- Lizano 0000331.pdf
- Lizano 0000334.pdf
- Lizano 0000336.pdf
- Lizano 0000338.pdf
- Lizano 0000339.pdf

- Lizano 0000342.pdf
- S. Lizano Deposition Transcript with Subject Matter Index.pdf
- 07.17.23 Sansanee Lizano Declaration Page - executed.pdf
- 2023.07.14 Sansanee Lizano's Amended First Written Discovery Responses.pdf
- 2023.07.17 Sansanee Lizano 1st Responses.pdf
- 2023.07.17 Sansanee Lizano Declaration Page - executed 2.pdf
- 2023.07.17 Sansanee Lizano Declaration Page - executed.pdf
- 2023.07.17 Sansanee Lizano First Written Discovery Responses.pdf
- 2023.08.04 Sansanee Lizano's Amended First Written Discovery Responses Executed.pdf
- 2023.09.08 New Indy HIPAA - S Lizano - Executed.pdf
- 2023.09.08 Sansanee Lizano Declaration Page.pdf
- 2023.09.07 Sansanee Lizano's Supplemental Written Discovery Responses 9.7.23.pdf
- 2023.09.08 Sansanee Lizano's Supplemental Written Discovery Responses 9.7.23 (redlined).pdf
- Michael Shane Nickell Deposition Transcript with Subject Matter Index.pdf
- Nickell, S\_Pineville Dentistry\_0001-0011, 1.pdf
- 2023.07.17 Shane Nickell First Written Discovery Responses.pdf
- 2023.09.07 New Indy HIPAA-Nickell Shane- Executed.pdf
- Tracie Nickell Deposition Transcript with Subject Matter Index.pdf
- 2022.11.02 Tracie Nickell's Responses to Defendants First Written Discovery.pdf
- 2023.02.17 Plaintiff Tracie Nickell's Supplemental Responses to Defendants First Written Discovery.pdf
- 2023.03.17 Tracie Nickell Answers to Defs' Second Discovery.pdf
- Tracie Nickell Answers to Defs' Second Discovery.pdf
- Nickell 0000058.pdf
- A. Swager Medical Questionnaire.pdf
- Amanda Swager Deposition Transcript with Subject Matter Index.pdf
- 2022.11.02 Amanda Swager's Responses to Defendants' First Written Discovery.pdf
- 2023.02.17 Plaintiff Amanda Swager's Supplemental Answers to Defendants First Written Discovery.pdf
- 2023.03.17 Amanda Swager Answers to Defs' Second Discovery.pdf
- 2023.09.07 New Indy HIPAA - A Swager - Executed.pdf
- Amanda Swager Answers to Defs' Second Discovery.pdf
- Swager 0000006.pdf
- Swager 0000008.pdf
- Swager 0000010.pdf

- Swager 0000013.pdf
- Swager 0000015.pdf
- Swager 0000016pdf
- Swager 0000018.pdf
- S. Swager Medical Questionnaire.pdf
- Shara Swager Deposition Transcript with Subject Matter Index.pdf
- 2023.07.17 Shara Swager First Written Discovery Responses.pdf
- Swager 0000088.pdf
- Kenny White Deposition Transcript with Subject Matter Index.pdf
- 2022.11.02 Kenny White's Responses to Defendants' First Written Discovery.pdf
- 2023.03.17 Kenny White Answers to Defs' Second Discovery.pdf
- 2023.09.07 New Indy HIPAA- White, Kenny-Executed.pdf
- 2023.09.08 Plaintiff Kenny White's Supplemental Answers to Defendant's First Written Discovery.pdf
- Kenny White Answers to Defs' Second Discovery.pdf
- White, K\_Charlotte Derm\_001-076.pdf

**Other Documents:**

- Complaint.pdf
- Current CAC.pdf
- EPA Clean Air Act Emergency Order.pdf
- EPA Filed Consent Decree.pdf
- DHEC Consent Order - Air.pdf
- DHEC Consent Order - Wastewater.pdf
- DHEC Order to Correct - Air.pdf
- CUMULATIVE H2S Data - Compiled for Morgan Lewis 2023-0519.xlsx
- Mill H2S Monitoring Data.xlsx
- Monitoring Data as of 8.2.23.xlsx
- New Indy Supplemental Community Monitoring Data\_2023-0811.xlsx
- 09.11.2023 Ltr re Service of Expert Reports.pdf
- 156 - Plaintiffs' ID of Expert Witnesses.pdf
- Expert Report of Allison Hecht, Ph.D..pdf
- Expert Report of Deborah Barsotti, Ph.D..pdf
- Expert Report of Harold Palevsky, M.D..pdf
- Expert Report of William Fee, M.D..pdf

- Expert Report of William Meggs, M.D., Ph.D..pdf